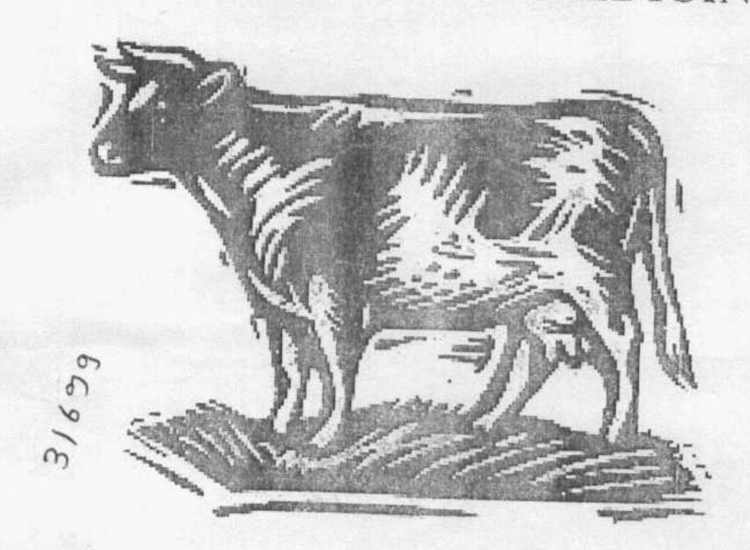
د/فتحي ناجي سليمان
الفرقة الخامسة

BENHA UNIVERSITY
COLLEGE OF VETERINARY MEDICINE
DEPARTMENT OF ANIMAL MEDICINE

LARGE RUMINANT MEDICINE



BY

PROFESSOR DOCTOR

Hussam El- Din M. El –Attar

PRESIDENT OF BENHA UNIVERSITY

PROFESSOR OF ANIMAL MEDICINE

DOCTOR
YASSEN M. ABDEL-RAOF
ASSISTANT PROFESSOR OF ANIMAL MEDICINE
BENHA UNIVERSITY

DOCTOR
MOHAMED M.GHANEM
LECTURER OF ANIMAL MEDICINE
BENHA UNIVERSITY

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DISEASES OF DIGESTIVE SYSTEM

STOMATITIS

Case: A calf or a cow or may be a group of them showing drooling of saliva, dullness and may be varying degrees of loss of appetite.

Definition :

Inflammation of the mucosa of the oral cavity including glossitis, palatitis and gingivitis caused by physical, chemical or infectious causes and characterized by profuse drooling of saliva, smacking of lips and a degree of anorexia

Causes:

The causes may be primary (physical and infectious causes) or secondary.

1-Primary causes:

A) Physical causes:

1- Sharp owns and spines of plants.

2- Fault use or vigorous use of stomach tube.

3- Fault in molar teeth such as maloccluded teeth or sometimes sharpness of the teeth.

4- Fracture of the mandible due to strong trauma from outside.

B) infectious causes:

1- Bacterial causes:

a) Necrotic stomatitis due to fusobactrium necrophorus.

b) Actinomycosis (lumpy jaw) and secondary infection.

c) Actionbacillosis (wooden tongue) due the actionbacillus lignieresi

2- Viral causes:

a) FMD

b) Cattle plague

C) Bovine virus diarrhea mucosal disease complex (BVD-MD).

d) Blue tongue in sheep.

symptoms:

A) General symptoms:

1- Varying degrees of anorexia may be partial or complete loss of appetite)

2- Drooling of saliva which may contain pus or shreds of epithelial cells. It may be few in amount causing froth at the commisures.

3- Painful mastication and smacking of the lips.

4- There may be fetid odour in the mouth due to bacterial invasion.

5- Enlargement of submaxillary lymph nodes.

6- May be fever in systemic cases.

7- local lesions:

A. Lacerated lesions on lips and tongue indicated traumatic cause.

B. Proliferative stomatitis: Small (less than 1 cm diameter), swollen, congested lesion on the tongue, buccal mucosa and palates which undergo ulceration in 3 days. It is usually occur in viral infection.

C. Papular stomatitis: Raised reddish papules (0.5-1 cm diameter) found on buccal mucosa and muzzle. It also may occur an the nostrils. It is another form of viral lesions and usually associated with other systemic reactions.

D. Pinpointed ulcers follows the papules occurs usually in cattle plague, and bovine virus diarrhoea mucosal disease, The ulcers coalesce together to form large denuded ulcers. These lesions are

found allover the mouth cavity, pharynx and oesophagus as well as the abdominal segments of GI tract. There is no vesicle formation. temperature may reach 40-41°C..

E. Vesicle formation which ruptured to leave ulcers. The vesicle filled with clear fluid. The temperature reachs 40-41°C but suddenly subside after the rupture of the vesicles.

F. Necrotic stomatitis: The lesions found on the mucosa of cheek. Also may be found on pharynx and larynx. The cheeks looked as swollen cheeked appearance.

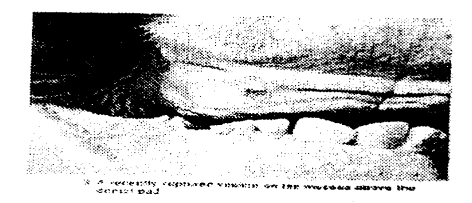
G. Wooden tongue of actinobacillosis: Abscess-like lesion in the tongue and may be the lymph node.



Hard swelling of the jaw (bovine actinomycosis)



Excessive salivation (stomatitis)




Ulcer at dental pad and erosion of the tongue in viral stomatitis

Diagnosis:

It depends on:

1- History:

Especially the presence or absence of fever, the duration of it, and its relation to the development of mouth lesions.

2- Symptoms:

General and local lesions. The development of the oral lesions. The relation of these oral lesions to other lesions or signs on other parts or systems in the body.

3- Laboratory diagnosis:

a) Culture from the lesions.

b) Serological tests.

Treatment:

A) Hygienic:

1- Isolation of affected animal.

2- Fresh clean water supply.

B) Medicated treatment:

I) General treatment:

1- Systemic antibiotics:

- Penicillin and tetracyclines are effective in necrotic type
- They also used in other types suspected to be caused by virus agent to avoid secondary invadors.

2-3 millions I-U. penicillin I/M daily. Or

2-3 gm tetracyclines I/V or I/M daily for 7-10 days.

2- Sulphonamides: Sulphathiazine 120 gm then 60 gm every 12 hrs.

3- Vaccination program: In case of diseases suspected to be caused by virus.

4- Antisera.

5- Iodides in actinomycosis and actinobuclosis (sod. iodide) 9 gm/100 Kg body weight in 20% solution givin I/V. also organic iodide may be given orally.

6- Isoniazid for actinomycosis and actinobacillosis 12-22 mg/kg body weight for 30 days.

II) Local treatment:

1- Antiseptic solutions to wash mouth cavity as:

a. Pot. permanganate 1:5000
b. Borax 1-2%
c. Boric acid 2%
d. Tannic acid 2%
e. Acriflavin 2%
f. Cupper sulphate 2%

2- Cauterizing agents to swab the lesion:

a. Silver nitrate 1%
b. Tr-iodine 1-2%
c. Sulphonamide in glycerin 0.5-1%

III) Radiation therapy:

It is used for treatment of actinomycosis and actinobacillosis. Never to be used before 30 days elapse after iodide therapy.

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PHARYNGITIS

Case:

A cow showing regurgitation of food few minutes after prehension. The history of the case reveals previous affection of respiratory system or oral cavity or may be vigorous use of stomach tube.

Definition:

Inflammation of the mucous membrane of the pharynx which is characterized by dysphagia, regurgitation of food few minutes after prehension, and may be cough.

Causes:

The causes are similar to those affecting the oral cavity. It includes:

I- Physical causes:

As the vigorous unskilled use of stomach tube which lacerates the mucosa.

II- Chemical causes:

As irritant vapour or irritant chemicals as undiluted irritant acids.

III- Infectious causes: such as:

1- Spheropherus necropherus which causes necrotic lesions on the buccal mucosa, pharyngeal and laryngeal mucosa.

2- Bovine virus diarrhoea-mucosal disease complex.

3- Rinderpest.

4- Actionbacillosis.

IV- By Extension,

From respiratory system affections or as a sequellae of stomatitis.

Symptoms:

The symptoms are belonging to both digestive and respiratory systems beside the general or systemic affection.

1- Dysphagia: The animal shows difficult swallowing.

2- Swallowed food is regurgitated few minutes After swallowing.

3- Increased salivation due to the irritant effect of inflammation.

4- Anorexia although the animal have the desire to eat. It try to eat but dysphagia, and regurgitation of food soon occur.

5- Nasal discharge which is usually watery or mucoid in nature.

6- Cough is frequent.

7- Pyrexia may occur.

8- Stiffness of neck.

9- Enlarged lymph nodes, either the retropharyngeal or the submaxillary

10- Palpation of the throat from outside reveals pain, hotness and swilling,

11- The use of laryngoscope also either necrotic foci or inflammatory spots of the mucosa.



Regurgitation of food



Extension of head and neck

Diagnosis:

It depends on:

1- History especially for the physical causes and those by extension.

2- Symptoms especially the time elapsed for regurgitation of swallowed food, dysphagia , and cough

3- Confirmative diagnosis by using the laryngoscope.

Treatment:

I- Hygenic treatment:

Prevent irritation by gases or smokes.

II- Supportive treatment:

Fluid therapy as Dextrose saline to compensate loss of natural feeding.

III- Medicated treatment:

(A) Antibacterial drugs to combate microorganisms:

1- Sulphadimidine sodium.

2- Suphathiazole.

120 gm orally then 60 gm after 12 hrs. and every 12 hours for 5 days.

3- Oxytetracycline 2-3 gm daily by mouth

4- Ampicillin:

2-3 gm daily for 5 days.

(B) Expectorant.

1- Rx.

Pot chlorate	8 g
Amm. carb.	16 g
camphor	4 g
Treacle	30 ml

MFT Dev. 6 doses one every 6 hrs.

2- Bronchiase I amp.

I/M every 2nd day.

(C) Medicated steam inhalation:

30 gm thymol or creoline on boiling water.

(D) Demulcents

(E) Analgesies and local anaesthetic.

CHOKE (OESOPHAGEAL OBSTRUCTION) IN CATTLE

Anatomical view of oesophagus in cattle:

It is a musculomemberanous tube which extends from the pharynx to the stomach. it is located medially to the trachea at its origin but at the 4th cervical vertebra. It is located on the left side of the trachea. Histologically it consists of mucosa which occurs in folds, submucosa (which contains elastic fibres, submucosal glands), the muscular layer (which consists of inner circular and outer longitudinal muscles), and the adventitia or fascia.

Definition:

Acute oesophageal obstruction usually occur in the cervical part of the oesophagus characterized by tympany, salivation and dysphagia.

Causes:

(A) Foreign body:

Any forign body may be engulfed with the food due to the type of prehension.

(B) Solid subjects:

Usually food but not completely masticated such as apple, orange, turnips, beets of sugar can, ear of corn and potatoes.

Pathogenesis:

Obstructed oesophagus may cause:

A- Prevent eructation which leads to tympany.

B- Prevents passage of food which leads to dysphagia and regurgitation of food.

C- May cause irritation which lead to restlessness and salivation.

Symptoms:

1- Sudden acute tympany with varying according to the degree of obstruction.

2- Profuse salivation.

3- Extended head and neck.

4- Continuous chewing movement.

5- Nervous restlessness.

6- Passage of stomach tube is impossible.

7- External swelling at the jugular furrow.

8- Regurgitation of food soon after swallowing.


Oesophageal obstruction

Diagnosis:

1- History:

- Sudden onset of tympany.

-Type of food.

2- Symptoms.

3- Confirmative diagnosis:

- Palpation of foreign body from outside.
- Passage of stomach tube to locate the site of obstruction.
- Free pharynx.

Treatment:

A- Hygienic treatment.

1- Prevent food intake.

2- Excess water supply.

B- Supportive:

Parental feeding.

C- Medicated treatment:

1- Sedatives to control pain:

a) Rumpon

b) Zylocain.

2- Tranquilizers as chloralhydrate (30 gm in 500 mL water by stomach tube or 30 gm in capsule.

3- Relief tympany: by using trocar and cannula.

4- Manual extraction of the foreign body:

a- Massage of the cervical part to activate peristalsis that relief spontaneously.

b- Massage upward and get-ride of the foreign body from mouth cavity either by fingers or by forceps.

c- Gentle knock of the foreign body by using stomach tube to push it toward rumen.

5- Surgically: rumenotomy.

DISEASES OF THE STOMACH

Anatomical and physiological introduction:

The stomach occupies about 75% of the abdominal cavity. It consists of four compartments:

1- Rumen:-It is the largest compartment of them.

-It forms 70-80% of them.

-It occupies all of the left side of abdomen

-It could be examined through the left flank.

-It capacity may approximate up to 60-80 gallons of water.

2- Reticulum: it follows the rumen and forms 7-10% of the stomachs. It lies anterior to 'the xiphoid cartilage.

3- Omasum: It follows the reticulum and forms 5% of the stomachs.

- It lies right to the medium plan opposite to the 1-8th rib and rest over the abomasum. Its left surface is in contact with the abomasum and rumen while its right surface is incontact with the diaphragm, liver and abdominal wall. It has dorsolateral folds.

4- Abomasum: - It is the true part of stomach .

-It resembles the stomach of monogastric animals.

-It forms 8-10% of the stomachs.

-It lies on the abdominal floor on the right side of the body. Anteriorly it is related (wide end) to the reticulum above the xiphoid cartilage and posteriorly it ends (narrow end) opposite to the 9 or 10th rib in the pylorus. greater curvature is incontact with the abdominal wall while the lesser one carry the omasum.

Function of the stomach

1- Bacterial digestion and fermentation by the rumen microfauna and microfauna on the ingested material to produce Volatile fatty acids (VFA)- formed of 70% acetic, 15% propionic, and 10% butyric fatty acids carbon dioxide, methan and other derivatives of amino acids deamination (such as amins and histamines).

2- Physical maceration by movement. These two main functions are interdependent so that rumenal movement is used as an index of digestive function.

3- Enzymatic digestion of food by the digestive juice of abomasum

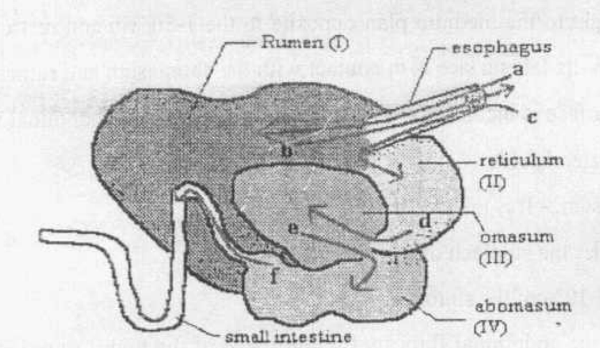
The layers of food content in rumen:

1- Free gas layer.

2- Fluid, food suspended particles, and air bubbles layer.

3- heavy and coarse particle layer which is the most down layer.

Gas
Mixed
Heavy



Normal flow of food from esophagus to stomach. The food is ingested (a) and fermented in the rumen. Larger particles are regurgitated (b) re-masticated and re-ingested (c). Sufficiently fine particles are passed to reticulum (d) and processed successively in the omasum (e) and enzymatically digested in the abomasum (f) before passing to small intestine.

Types of ruminal movements:

1- Eructation movement:

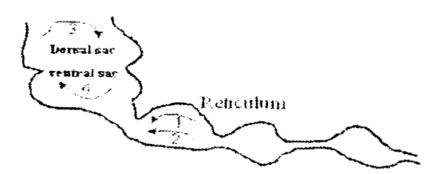
It occurs by the contraction of the dorsal Sac of the rumen (air containing layer of the rumen).

2- Rumination movement:

it occurs by deep inspiratory effort to creative pressure in the thorax followed by escaping of the reticular fluid to the cardia then to the oesophagus which stimulates antipersistaltic movement. The anti-peristaltic movement direct the fluid and its containing food material toward the pharynx and remastication takes place. Rumination is stimulated by the coarse fibres found in rumen and reticulum.

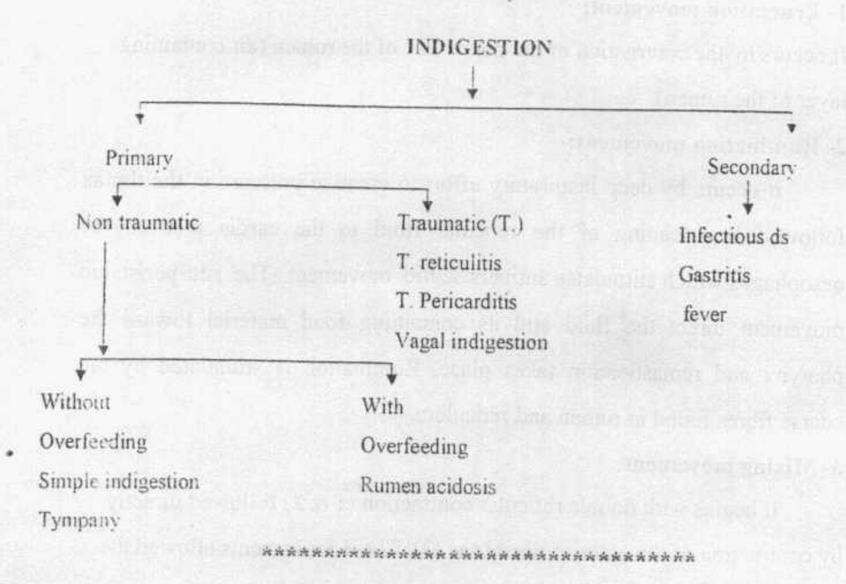
3- Mixing movement:

It begins with double reticular contraction (1 & 2) followed directly by contraction of the anterior dorsal sac (3). These movements allowed the fluid in the recticulum to return back to the rumen.



The dorsal sac contraction makes a sort of rotation in the rumen, this rotation is completed by contraction of the ventral sac (4). The end result of all these contractions is the maceration of the food particles found in the rumen.

The main disturbance in the ruminant stomach is related to either the defect in the microbial digestion or the mixing movement which leads to the so called "indigestion" as a general name.



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SIMPLE INDIGESTION

Case:

The case admitted with complaint of decreased food intake with mild decrease in milk production. The case may shows lack of rumination or even some sort of constipation.

Definition:

A case caused by atony of the rumen or even the forestomach and is characterized by anorexia, lack of ruminal movement, and constipation.

Causes :

It is usually occurs in dairy cattle more than in beef cattle.

1- Dietary causes:

(a) Indigestible roughage especially with low protein in take.

(b) Mouldy, hot frozen food.

(c) Gross overfeeding especially with groins and concentrates.

(d) Sudden change to wheat or barely.

2- Limitation to available drinking water which usually occur in dry season.

3- Prolonged administration with sulfonamides and antibiotics.

Pathogenesis :

1- Primary atony is difficult to explain but the change in PH or the absorption of toxic amids or amins may be the cause.

2- Grains and fermentative reaction or carbohydrate results in increase acid production which change the optimum PH of rumen for the normal microflora (normal pH 6.8).

3- The high protein, legumes and concentrates cause increase in alkalinity This change in PH to either sides depress certain kinds of miceflora or micofauna. This result will cause decrease in the motility.

4- Damage food either affect the PH or produce toxic substance that cause atony of rumen.

5- Grass over feeding may cause physical interference with rumen motility.

6- The end result of the failure of the function of the forestomach is the decrease production of volatile fatty acids with consequent effect on the milk fat and quantity of milk production.

7-Anorexia is produced due to the absence of the rumen contraction which resembles hunger contraction in the monogestric species.

Symptoms :

1- Partial or complete anorexia.

2- Depression and may be dullness.

3- Delayed or depressed ruminal movement either by palpation or auscultation.

4- Constipation followed usually by diarrhea.

5- Tympany may occur due to damage food or putrefactive effect on food.

6- Slight decrease in milk yield.

7- Lowered turnover rate with the consequent loss of weight gain.

Laboratory examination:

1-Sediment activity test

Aspirate ruminal fluid and filtrate it to remove coarse particles. Put the filtered ruminal fluid in a glass vessel. Add a straw particle to it., put in incubator at 37°C and watch the time allowed to it to sink and float again. Normal sediment floatation time is 3-9 minutes according to the type of feed. Delayed time indicate inactivity.

2- Cellulose digestion test :

Put the filtered ruminal fluid in a glass peaker. Put a cotton thread (cellulose matter) ended with glass beeds in the baker, put in the incubator at 37°C. Watch the time need for complete digestion of the cotton thread (cutting of the thread).Normal time is 24-30 hours. Delayed time more than 30 hours indicate abnormal in the activity of ruminal juce.

3- Microscopical examination:

Put one drop of ruminal juice on a glass slide add one drop of lugol's iodine. Cover with cover slide and examined directly under microscope for the activity of the infuseurea .

Diagnosis:

It depends on:

1- The history: regarding the amount and type of food of carbohydrate type or protein typr. The deform of food weather intake or grounded and the presence of mould or decomposition.

2- Symptoms.

3- Lab.diagonsis.

4- Differential diagnosis: Must be differentiated from:

a- Acetonemia by the history of presences of ketogenic fod and lab. exam for ketone in blood. urine and milk (Rother's Test) & the time of calving.

b- Traumatic reticulopertionitis by the history of recurrent gastric troubles and the pain test.

c- Abomasal displacement by the tinkling sound of ausculation and recent of parturition.

Treatment :

I- Hygenic treatment:

1- Allow the animal to thoroughly to exclude moulds or other decomposing substance.

2- Examine food materials thoroughly to exclude moulds or other decomposing substance.

3- Correct the amount of carbohydrate or the carbohydrate of the ration (such as grains or legumes and urea %)

II- Medicated treatment :

Usually symptomatic to restore both rumen movement and microbial activity.

A- Rumen tonic drug:

These types of treatment is planned to restore the rumen movement.

These drugs used have an active principles as follow:

1- Tarter emetics 10 – 12 gm orally in diluted form to avoid the irritant effect of the drug.

2- Parasympathomimetic (2.5 mg/100 lb.). It is used in small doses and short intervals to obtain their nicotinic effect such as cerbamyl choline HCL, which act on smooth muscle fibers to increase contractility of the gastrointestinal tract.

- Physostigmin.
- Neostigmine.

B- Purgatives:

1- mineral oil (paraffin oil) It act as mechanical which envelop the food particles and lubricate. Its passage in the G.I.T.

2- Saline purgative: It acts by increase the water content of the intestine and consequently stimuli the peristalsis via the afferent and efferent fibers of the vagus nerve such as: Epsom salt (1 – 2) orally as drench).

Anthraquinone (8 – 15 gm or 0.5 gm orally as French).

C- Stomachics:

It acts by increasing the appetite via the henger contraction so that stimulate the contractility of the compound stomach .

- Strychnine preparation is used orally up to 65mg as a single dose such as:
  - Stomavetic
  - Bykodigest
  - Vapicodigest
  - Bremadigest:

-Gastric stimulants:

R/ Amm. Carbonate 16 gm

Oil of turpentine 30 cc

Linseed oil or H_2o to 1litre

Prepared 3 times with 12 hrs interval if necessary

D- Adjustment of PH present:

Correction the ruminal PH by using substances opposite to the PH present.

I- In case of increase acidity: We can use substances of alkaline radicale such as: Magnesium hydroxide (Milk of magnesia) 500 gm dissolved in water. Sodium bicarbonate (200 – 500 gm) is also used.

II- In case of increase alkalinity: We can use substances of acid-radile such as:

- Vinger: 50 100ml in one litre of water.
- Diluted HCL.

E- Rumen transplantation:

Reconstitute the rumen flora specially if the PH was changed. We can obtain rumen flora from abattoir or from healthy living animals (1-2 litres) Also, commercial rumen products or commercial "instant roughage".

F- Antihistaminics: to correct the harmful effect of histamine produced by the improper fermentative digestion Avil: 3 amp I/M, IV – Allercur: 3 amp

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RUMINAL IMPACTION
GRAIN POLSONING – GRAIN ENGORGMENT
ACUTE RUMINAL ACLDOSIS

CASES:

1- A dairy animal admitted to the clinic with a history of accidental release to a sac of flower or grounded maize or wheat with the subsequent symptoms of complete anorexia. complete subsequent symptoms of complete anorexia. complete suppression of rumination anorexia complete suppression of rumination and drop of milk yield.

2- A beef calf admitted to the clinic with the complaint of loss of appetite and rumination as well as some nervous manifestations and lameness. The history of feeding material nitrogenous substance (urea).

3- A beef calf admitted to the clinic with the complaint of loss of appetite and rumination. The past history indicated that the calf had been puted in a highly concentrate for the final finishing of marketing

Definition :

It is a condition in which the animal ingested large amount of highly fermentable carbohydrate resulting in high amount of lactic acid formation or ingestion of large amount of ration containing large amount of non-protein nitrogenous compound with the resulting of high ammonia. Both conditions will cause blindness, complete ruminal stasis, anorexia and toxemia.

Causes :

(A) Ingestion of large amount of whole or ground grains before proper period of adjustification which may be predisposed by:

1- Fresh harvested grains are more toxic.

2- Animals break into field or ripegrain corn may engorge themselves more than those intable feeding.

3- Sudden change from higher grains to wheat and barely.

The normal dose of grains was 5 kg per cow daily and after accustoming may reach up to 20 hg

The lethal dose rate of grain rations are:

- for sheep 60 – 80 gm per kg b. wt
- for cattle 20 gm per kg b. wt

(B) Ingestion of large amount of feeds containing non – protein nitrogenous material (urea).

The urea percent must not exceed 3% of the amount of the ration feed to animal.

Pathogenesis:

1- High carbohydrates in ration will be acid upon by the gram positive cool (mainly Strept bovis) to ferment them into high amount of lactic acid.

If the amount of lactic acid in rumen exceed 3% it will produce high osmotic pressure which consequently withdraw fluid to the rumen.

The withdrawal of fluids results in a case of tissue dehydration, blood haemoconcentration, and anuria.

The increased amount of lactic acid with the subsequent lowering of the pH will destroy the normal microflora so that the final product of normal fermentation (the volatile fatty acids) is ceased. The change of the PH of the rumen had a direct effect on the rumen motility with the resultant verying fegrees of ruminal stasis. PH of 4.5-5 cause incomplete ruminal stasis, while pH lower than 4.5 results in complete ruminal stasis

2- High amount of non-protein nitrogenous compound (urea) will be acted upon by proteus microorganisms with the resultant of ammonia. Toward the alkaline side. This will lead to suppression of the normal microflora and the degrees of volatile fatty acids.

3- Death may occur due to dehydration or haemoconce-ntration.

The state of acidosis in the rumen lead to the release of the alkaline raddicle of he blood to the rumen with subsequent scidic condition of blood (academia). Acidemia may lead to nervous manifestation death again.

On the other hand alkalaemia will occur in case of rumen alkalosis with the subsequent nervous manifestation and death also.

4-Production of histamine in the rumen to a level more than 70 per ml will produce a case of depression and polypnoea.

5- Increase acidity affect the rumen mucosa and produce chemical ruminitis. Invasion of the inflamed mucosa by bacter and fungi results in metastatic abscesses. necrosis and gangrene and acute peritonitis.

Symptoms:

The speed of onset or these symptoms depend upon the nature of the feed. while its severity depend upon the amount of feed.

1- Depression with loss of alert condition of the animal and lowering of the head.

2- Anorexia with complete loss of ruminator .

3- Abdominal pain .

4- Distention of the abdomen and may be slight tympany

5- Staggering in gait.

6- Constipation which may be followed by sever diarrhea.

7- Increase pulse and respiration rate while temperature may be either normal or subnormal with the associated recumbancy and cold extremities.

8- Laminitis may occur.

9- physical examination reveal firm doughy content of the rumen by palpation of the left flank while auscultation reveals complete ruminal stasis with some gurgling sounds of gases.

Prognosis :

It is unfavourabe when the symptoms had rapid onset, subnormal temperature or when the history indicated finally grounded feed material.

It is unfavourable if improvement occur rapidly or when the rise of body temperature and passage of amount of faeces.

Laboratory tests:

(A) urine sample:

1- pH: Normally, it is 8. In severe cases down to 5.

2- Colour: concentrated.

3- Volume: Decrease in amount (oliguria).

4-Some proteinuria.

(B) Blood :

1- PCV: It is increased from normal of 30-32% to reach 50-60% in case of haemoconcentration.

2- Increase of blood lactate and inorganic phosphate.

3- Blood pH: To detect either academia or alkalaemia (normal pH of blood 7-7.2).

(C) Rumenal fluid:

1- To detect pH (normal pH of rumen 6.8).

2- Flora. Gram negative organisms may be replaced by Gram positive Streptococci.

3- Microflora. Disappearance of Enfusuria.

Diagnosis :

It depends on :

1- The history

2- Clinical symptoms.

3- Laboratory diagnosis.

Treatment :

(A) Hygienic treatment:

1- Supplement the grain rations with large amount of hay.

2- Withheld water supply to the limited amount.

3- Nursing of recumbent animals by changing its position and adding good bedding of Rice Straw.

(B) Supportive treatment:

intravenous injection of 500ml physiological saline.

(C) Medicated treatment:

It is plained to:

I- To evacuate rumen by :

1- oil purgative (1-2 litres of mineral oil or vegetable oil orally).

N.B: Avoid saline purgative as much as possible

2- Surgically by rumentomy.

II- Stop the multiplication of microorganisms by:

1- using antibiotics as oxyteracycline powder (2-3 gm orally)

N.B: Avoid chlorotetracycline orally as they destroyed by microorganisms.
Sulphadimidine or sulphaguanidine (120 150-gm) orally followed by 60 after 12 hours and avery 12 hours

III- Correct pH by:

a- Lime stone (250 – 400 gm) then followed by 120 gm after 12 hours and every 12 hours.

b- Magenium hydroxide or magnesium bicarbonate (250 -400gm) then followed by 120 gm after 12 hours and every 12 hours.

c- Sodium bicarbonate orally 200 – 500gm and I/V as 2% solution (500ml).

d- Calcium borogluconate 2% solution I/V (200 – 400 ml).

IV- Treatment dehydration:

In case of academia, saline bicarbonate electrolyte therapy (1-2 litres I/V).

Lactate Ringer in case of alkalaemia.

V- Antihistaminics: Avil or allercur

Prevention:

The case may be avoided on large scale by: gradual changing of ration e.g. begin with 25% grains plus 75% roughage.

Gradually change this ratio over one moth to be 75% grains plus 25% roughage (gradual adjustification).

Ad[illegible] unded lime stone and sodium bicarbonate (equal amounts to be up to [illegible] the ration).

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RUMINAL TYMPANY OR BLOAT

Case:

A cow or, buffaloe or sheep or goat with a complaint of sudden gaseous distension of the abdomen. The history of feeding reveals leguminous succulent feed. The cow showed marked dyspnoea, restlessness and sever loss of production.

Definition:

A case of over distension of the rumen and reticulum with gases which may be either free separated gas (free gas tympany) or mixed with the ingesta (frothy tympany). It is characterized by severe loss of production, over distension of the flank with gases and may be death.

Causes:

The cause may be primary which is usually frothy in character or secondary which is usually of free gas type.

A) Primary causes:

The primary causes will direct to the type of food or the susceptibility of the animal.

1- Grassing instead of poor pasture.

2- Feeding legumes in the preblooming stage.

3- Feeding on tympanogenic feeds such as cabbage, cereal crops, or leguminous vegetable crops as peas or beans.

4- Grazing on wet pastures with dew as occur early in the morning.

5- Animals vary in susceptibility for forming frothiness.

B) Secondary tympany:

These are directed to causes or factors, which interfere with getting ride of normally, produced gases as:

1- Choke or oesophageal obstruction.

2- Obstruction of the cardia by carcinoma or enlarged lymph nodes.

Pathogenesis :

A) Frothy type :

Saponins and pectins concentrated on the rumen fluid lowering its surface tension.

1- Saponins and pectins will increase both the viscosity of the normally formally formed air bubbles can not easily either reach the surface or to erupt at this surface.

2- Increase protein solubility of piants will increase the slime formation and consequently increase the viscosity of ingesta and so increased foam formation (The main soluble protein is 18 – s protein .

3- Legumes sink down to the lower part of rumen and they produce large amounts, will easy to reach the surface. On the other hand legumes are rich in organic acids that react with the bicarbonate buffer of rumen and release large amounts of co_2

4- L[illegible]ered rumen PH (5.4) favours the growth of encapsulated bacter and conse[illegible]ntly increase viscosity. At this PH, also, mucinolytic bacteria grow and destroy salive much which is a antifoaming agent.

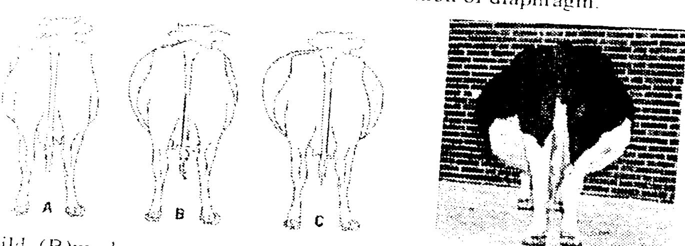
B) Free gas tympany:

The normal formed gas can not eructated by the usual way.

Symptoms:

1- The animal show restlessness and discomfort. The animal may lie down and rise again

2- Sudden and rapid distention of the left flank and all the abdomen. This animal may show great tendency for eructation of diaphragm.



(A)Mild. (B)moderate and (C) severe distension of left flank region



Distended abdomen (Acute tympany)

3- Increase respiratory rate. The appetite is reduced as well as the milk production.

4-Abdominal pain may be manifested by looking of the flank or by the aimless movement of the animal. The animal may recumbent bulged eyes or protruded tongue in this case death may occur with 10 minutes.

5- By percussion reveals tympanic sound auscultation the rumenal movement is completely stopped (complete stasis).

6- passage of stomach tubes allow the free gas layers to get out of rumen. This free gas layers to very small in case of frothy tympany followed by obstruction of the stomach rube orifice by the ingesta.

Lab. Diagnosis: not necessary.

Diagnosis:

By history especially the sudden onset and the type of feeding symptoms.

Differential diagnosis:

To differential between free and froth tympany using stomach tube.

Treatment:

A- **Hygienic treatment :**

The hygienic treatment is directed to help the animal get rid of the gas in the rumen by:

1- Stand the animal with his forelimbs in a level higher than hind limbs.

2- Put a stick in the mouth to keep it open.

3- Put a wood on the tongue to enhance eructation.

4- Rumen massage to help contractility.

5- To avoid recurrency of this case you must add 5kg hasy/head daily to the ration while the legumes must be less than 50%.

B- Medicated treatment:

The medicated treatment should be directed to change the frothy tympany to free gas tympany then get rid of this gas:

1- To reduce the stability (lowering the surface tension) by mechanical purgative as vegetable or mineral oil or we can supplement this treatment with 30 cm of turpentine oil, kerosein formaline as antifoam agents.

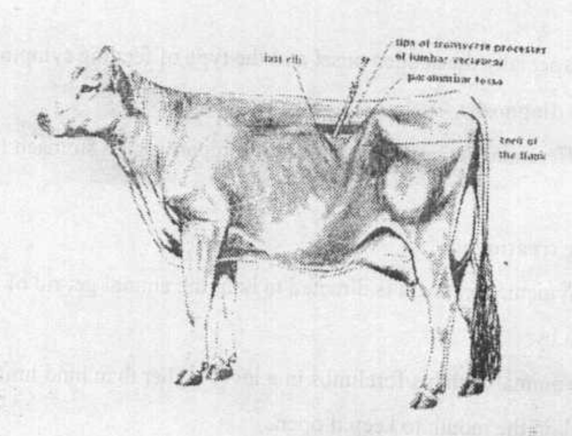
2- Can use antibiotics to suppress the microbial fermentative actions, after changing the frothy tympany into simple gas tympany use stomach tube to get raid of gases.

3-Antihistaminie should be administrate as AVIL 2-3 ampoules I.M. or I.V.

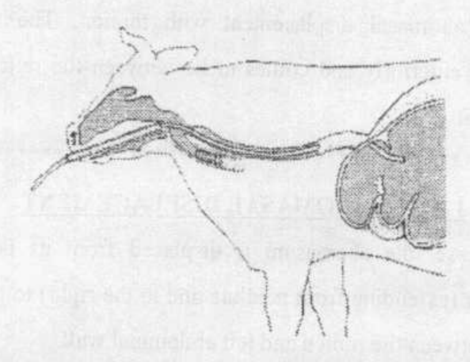
C-Surgical interference :

i) Rumonatomy.

ii) Trocar and canula



Trocar and cannula for emergency treatment of tympany

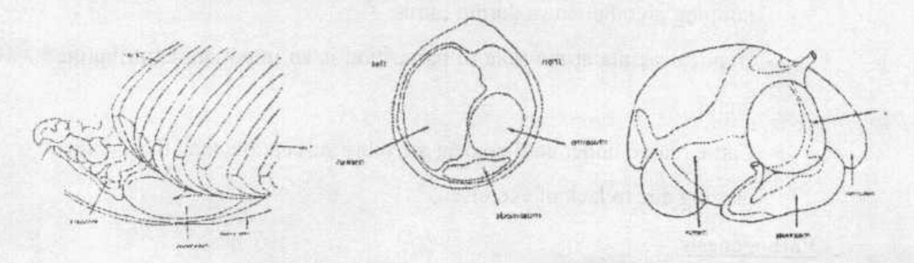


Passage of stomach tube helps gas to go out of rumen

ABOMASAL DISPLACEMENT

Anatomical and physiological aspect

- Abomasum is the fourth compartment of ruminant stomach, which is the true stomach responsible for the enzymatic digestion of food.
- The abomasum is localized at the right front abdominal floor between rib 7 and 13.



ABOMASAL DISPLACEMENT

The abomasal may be displaced from its normal anatomical position to either right, left or anterior situations leading to disturbance in digestive functions. Therefore, abomasal displacement may be:

1- Left abomasal displacement

2- Right abomasal displacement
3- Anterior abomasal displacement with torsion. The abomasum is displaced anteriorly and comes to lie between the reticulum and the diaphragm.

****

I- LEFT ABOMASAL DISPLACEMENT

In this case, the abomasum is displaced from its position at the abdominal floor (extending from medline and to the right) to the left side of the abdomen between the rumen and left abdominal wall.

Incidence, Occurrence and Predisposing factors

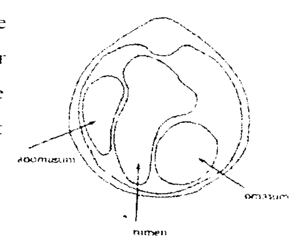
1- The disease occurs most commonly in heavy milk producing cows, specially after parturition (6 weeks post-parturient in 91 % of cases).
2- Cattle feeding heavy grains, such as corn and the roughage level is less than 17 % of the diet.
3- In some cases, the disease is associated with unusual activity, such as jumping on other cows during estrus.
4- Hypocalcaemia at the time of parturition is an important contributing factor
5- Cattle reared under confinement are more susceptible than those under grazing due to lack of exercise.

Pathogenesis

1- Heavy grain feeding increases the concentration of free volatile fatty acids in the abomasum, which inhibit its motility leading to abomasal atony. Consequently, the flow of ingesta from abomasum to duodenum is retarded and accumulated in the abomasum. Gas

production is increased, especially methane, leading to further distention following by displacement.

2- During the late stage of pregnancy, the rumen is lifted from the abdominal floor by the expanding, gravid uterus and the abomasum is pushed foreword and left under rumen. After parturition, the rumen subsides trapping the abomasum leading to displacement, specially when the abomasum is atonic.



Clinical signs:

Usually starts few days or a week after parturition

1- Inappetence, and sometimes-complete anorexia.

2- Marked drop of milk production.

3- Left abdominal distention, which gives the shape of slab-sided left abdomen (thick and flat).

4- The pulse, respiration and temperature are usually normal.

5- The feces are reduced in volume and softer than normal, but periods of profuse diarrhoea may occur.

6- Decreased frequency and intensity of ruminal movements.

7- Auscultation in the area below a line from the center of the left paralumber fossa to the left elbow reveals higher pitch tinkling or splashing sounds (abomasal sounds). These sounds occur as long as 15 minute a part.

8- Percussion over the area between the left 9^{th}-12^{th} intercostals space reveals high-pitched tympanic sounds.

9- Rectal examination revealed empty upper right abdomen.

10- Varying degrees of ketosis.

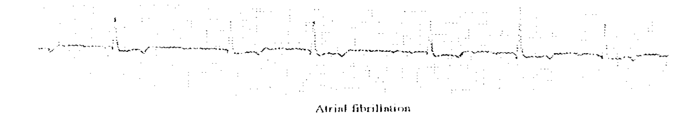
Diagnosis

I- Case History

II- Clinical signs

III- Physical examination.

IV- ECG: reveals paroxysmal atrial fibrillation due to metabolic alkalosis.



Atrial fibrillation

V- Differential diagnosis:

a. *Ketosis*: treatment of ketosis is permanent, while treatment of ketosis *secondary* to abomasal displacement is temporary and relaps usually occurs in a few days.

b. *Traumatic reticuloperitonitis*: is usually associated with fever and positive pain test (grunting sound)

c. *Vagal indigestion*: abdominal distension due to distended rumen with or without an enlarged abomasum usually occurs before parturition

d. *Diaphragmatic hernia*: characterized by chronic ruminal tympany and abdominal distention

Treatment

1- Rolling of the animal: the animal is cast and laid on the back, then rolled vigorously to the right and the rolling stops abruptly in the hope that the abomasum will free itself and replaced to normal position. Starvation and restriction of fluid for 2 days before rolling is advisable.
2- Violent exercise and transport over bumpy (uneven) roads occasional cause spontaneous recovery.
3- Surgical interference
4- Treatment of the secondary ketosis by parenteral glucose and oral propylene glycol.

Prevention

1- Ensure that calculated total crude fiber in the ration exceeds 17 percent.
2- Make all feed changes near calving time slowly, especially any increase in grain or other concentrates.
3- Give cows plenty of time to reach peak production.
4- Encourage exercise.

****

RIGHT ABOMASAL DISPLACEMENT (ABOMASAL DILATATION)

Definition

It is a subacute disease of mature cows occurs a few weeks after calving and characterized clinically by inappetence, depression, dehydration and gradual distention of the right side of the abdomen due to accumulation of gas and fluid in the abomasum.

Etiology, incidence, occurrence and predisposing factors

1- The disease mostly occurs 3-4 weeks after calving.
2- Calves may be accidentally affected.
3- The disease mostly occurs during winter seasons because of the heavy grain feeding indoors for milk production.
4- Atony of the abomasum is the major cause of dilatation, but obstruction at pylorus could also a contributing factor.
The incidence of right abomasal displacement is 10%, but is more fatal then left type (incidence 90 %)

Etiology and pathogenesis

A- Dilatation stage

1- Atony of the abomasum occurs initially resulting in accumulation of fluid and gas leading to gradual distention and displacement in a caudal direction on the right side.
2- During the dilatation phase, there is continuous secretion of hydrochloric acid, sodium chloride, and potassium into abomasum leading to dehydration, metabolic alkalosis, hypochloremia and hypokalemia.

B- Torsion stage

After the dilatation stage, the abomasum may twist in clockwise or anticlockwise direction, causing signs of acute obstruction and local circulatory impairment and ischemic necrosis of abomasum.

Clinical signs

1- Inappetence, poor milk production.
2- Depression, dehydration, and excessive thirst.
3- Muscular weakness (hypokalemia)

4- The mucous membranes are pale and muddy.

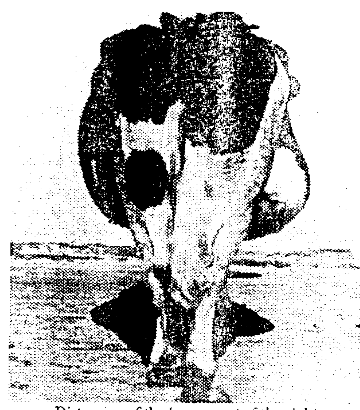
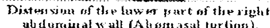
5- The feces is dark, soft and scanty.

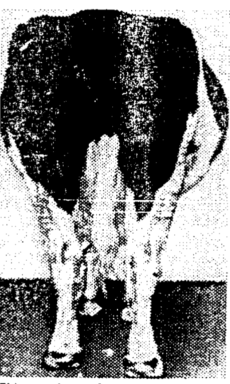
6- Distention of the right side of abdomen as abomasum is displaced between liver and right abdominal wall and can be palpated under the right costal arch.

   a. Palpation reveals fluid-splashing sound and distended abomasum with tense wall

   b. Percussion produces gassy pings (tympanitic resonance)

7- If torsion occurs, there is sudden onset of abdominal pain and kicking at the abdomen with sever depression. Death may occur after 48-96 hour due to shock and dehydration.



Distension of the lower part of the right abdominal wall (Abomasal tortion).


Distension of the right flank region (Abomasal tortion).

Diagnosis

I- Case history

II- Clinical signs

III- Physical examination.

IV- Lab Diagnosis

a. Hemoconcentration (increased PCV and plasma proteins)

b. Metabolic alkalosis, hypochloremia and hypokalemia.

c. Paradoxic anuria may be present due to excretion of acid by kidney in response to severe potassium depletion

V- Differential diagnosis:

a. Abomasal impaction: palpation reveals firm doughy mass located on the lower aspect of the costal arch, at the floor of abdomen, whereas abomasal distention can be palpated dorsally close to the right paralumbar fossa

b- Vagal indigestion:

Treatment

Prognosis is favorable if treatment is initiated a few days after the onset of clinical signs:

A- Mild cases (abomasal dilatation only):

1- Medical treatment started with IV injection of 500-1000 ml CBG 25 %.

2- Provide good quality hay and avoid grains for 3-5 days.

3- Early treatment with fluids and electrolytes IV and orally often yield good results. The fluid therapy is essential to restore the motility of gastrointestinal tract, particularly abomasum.

4- Mineral oil (5-10 liters /day orally) and magnesium hydroxide (500 g orally for adult cow daily every 2 days) to evacuate the contents of abomasum, especially after correction of the fluid and electrolyte imbalance.

B- **Severe advanced cases and torsion**: surgical correction is indicated

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ANTERIOR ABOMASAL DISPLACEMENT

- The abomasum, or portion of it, is displaced anteriorly between the reticulum and diaphragm.
- The clinical syndrome is similar to the left abomasal displacement but clinical signs are mild.
- There is no interference with the blood supply to the trapped portion but the effects are only due to interference of digestion and movement of ingesta leading to a state of chronic inanition.
- Mild ketosis, hypochloremia and hypokalemia may develop due to continued secretion of hydrochloric acid into abomasum.
- Treatment: by surgical interference only.

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VAGAL INDIGESTION

Definition

Chronic intermittent abdominal distention due to lesion of the vagus nerve supply to the forestomachs and abomasum cause varying degrees of paralysis of stomach with delaying of passage of ingesta, distention, anorexia with passage of small quantity of soft pasty feces.

Etiology:

The vagus nerve runs along both sides of the esophagus and terminates in branches that innervate the forestomachs and abomasum.

1- Traumatic reticuloperitonitis is the commonest cause.

2- Inflammation and scar tissue lesion affecting the ventral branch of vagus nerve.

3- Actinobacillosis of rumen and reticulum is a common cause.

4- Involvement of vagus nerve in the thorax may occur due to enlargement of the lymph nodes by tuberculosis and lymphosarcoma or because of diaphragmatic hernia.

5- Inflammation or traumatic damage to the nerve can result from pharyngeal trauma or abscesses.

6- Perforating abomasal ulcer (Hardware disease) with diffuse peritonitis can interfere with outflow of ingesta from abomasum.

Pathogenesis

1- Involvement of vagus nerve branches in adhesion or inflammation leads to achalasia of the reticulo-omasal and the pyloric sphincters, although paralysis of the forestomach and abomasal walls also plays a part.

2- When there is achalasia (sustained contaction) of the reticulo-omasal sphincter, ingesta accumulates in the rumen. If the ruminal wall is atonic, the ingesta accumulates without bloat occurring; if it has normal motility, the ruminal wall responds to the distension by increased motility and the production of frothy bloat.

3- When there is achalasia of the pylorus, there is blockage of ingesta at this point and a syndrome of pyloric obstruction and abomasal impaction develops.

Clinical signs

Three clinical syndromes of Vagal indigestion are identified:

A- Ruminal distension and hypermotility.

B- Ruminal distension with atony

C- Pyloric obstruction and abomasal impaction.

The common clinical signs in all syndromes are:

1- Inappetence for several days.
2- Scant feces.
3- Abdominal distension due to ruminal enlargement and abomasal impaction.
4- No response to treatment.

A- Ruminal distension and hypermotility.

1- This syndrome is not particularly related to parturition and pregnancy.
2- Moderate to severe ruminal tympany although the animal is thin and not eating well (inappetence) for some times.
3- Apple-shaped abdomen (distension of rumen).
4- The rumen movement is forceful and continuous.
5- Rectal exam. reveals distension of the dorsal sac of the rumen, which is pushed back against the brim of the pelvis
6- The feces is normal or pasty but scanty.
7- Temp. is normal
8- Systolic heart murmur, which waxes and wanes with respiration.

B- Ruminal distension with atony

1- This type occurs most commonly in late pregnancy and may persist after calving.
2- Ruminal movement is seriously reduced or absent.
3- Abdominal distension with no response to purgative, lubricants or parasympathetics.
4- Persistent mild bloat.
5- No fever, no increase in heart rate, and no pain.
6- Rectal examination reveals distension of the rumen which blocks the pelvic inlet.

7- The animal loses weight rapidly, becomes very weak and even recumbent. At this stage, the heart rate is increased and the animal may die due to inanition.

C- Pyloric obstruction and abomasal impaction

the most serious form

1. Mostly occurs at late pregnancy.
2. Anorexia and scanty pasty feces.
3. Distension of abomasum due to impaction that can be detected by rectal palpation, where the distended abomasum is palpated at the abdominal floor. The abomasum is firm and not distended with gas or fluid. Palpation is impossible if the cow is pregnant.
4. No systemic reaction, except at the late stage, when the pulse rate is rapidly increased.
5. Ruminal movement is completely absent.
6. Death may occur due to rupture of abomasum.

Diagnosis

- I- History
- II- Clinical signs
- III- Lab. Findings:
  - a. Mild hypochloremia, hypokalemia and alkalosis.
  - b. Dehydration

Treatment

The prognosis of most cases is unfavorable

1- Some cases respond to fluid and electrolyte therapy for 3 days combined with oral administration of mineral oil (5-10).

2- In pregnant cows, fluid and electrolyte therapy is indicated near enough to term to induce parturition by dexamethasone (20 mg IM),

but the condition may recur in the next pregnancy.

3- Rumenotomy and emptying the rumen when there is ruminal hypermotility is indicated. A permanent ruminal fistula to permit the escape of gas may cause dramatic improvement.

4- Subcutaneous administration of calcium borogluconate is indicated in ruminal atony.

5- Slaughter the affected animal for meat is the most satisfactory procedure.

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TRAUMATIC RETICULOPERITONITIS (HARDWARE DISEASE)

Definition:

A form of indigestion that is caused by ingestion of sharp hard foreign body that result in inflammation of reticulum and peritoneum and characterized clinically by:

1- Abdominal pain.

2- Anorexia

3- Fever

4- Decrease in milk production

5- Complete rumen stasis

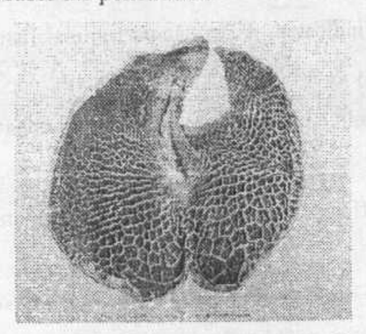
6- Recurrent tympany

Predisposing factors:

1- The diseases occurs in bovine only due to the method of prehension (prehension occurs by tongue only).

2- The anatomical structure of the reticulum, which is a funnel or flask shape facilitating penetration by foreign bodies.

3- Anatomical structure of reticular cells, which have a honey comb shape that facilitate holding the foreign body and then muscular contraction increases the penetration.



Honeycomb or hexagonal cells of reticulum

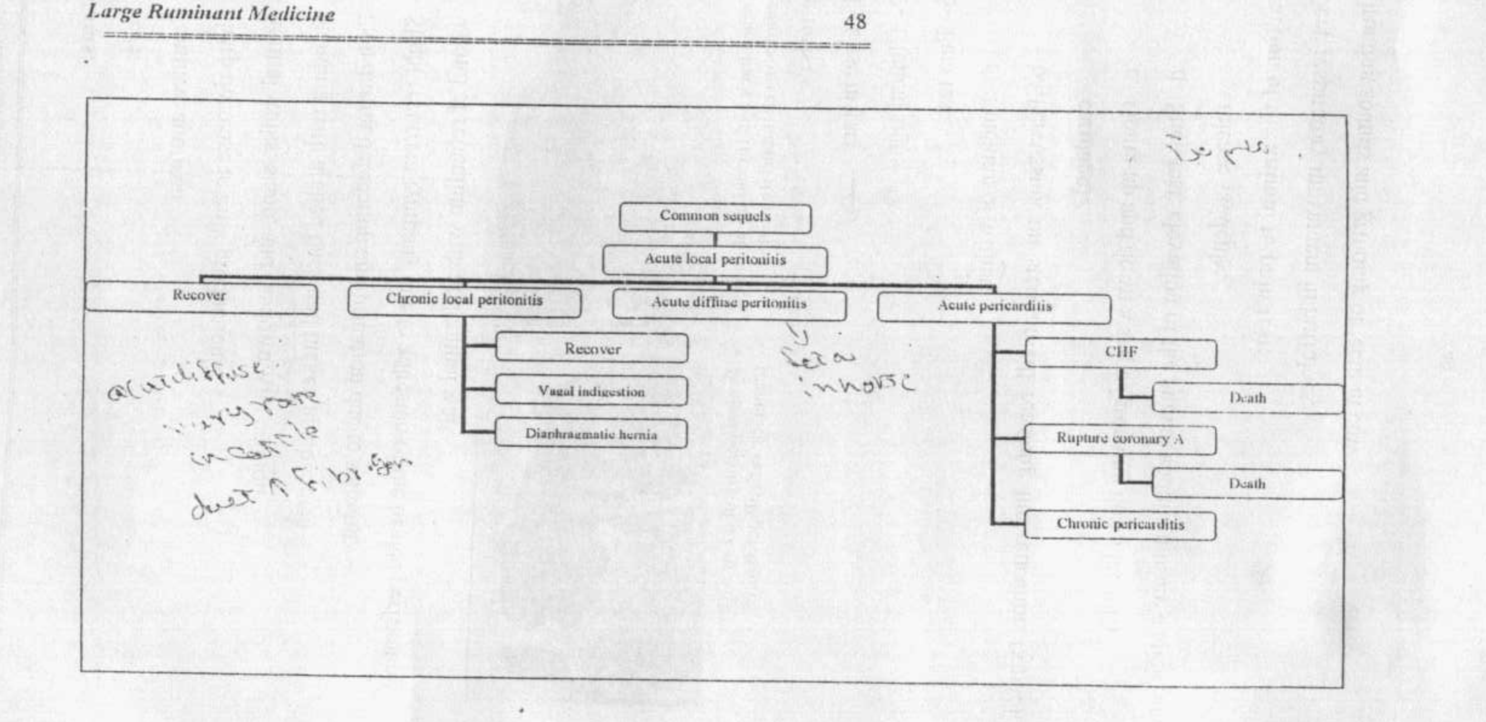
Etiology and pathogenesis:

1- Ingestion of sharp hard F.B. 48 % wire 38% nails, 14% others.

2- Symptoms mostly appear in late stage of pregnancy, after parturition, or constipation or transportation. due to contraction ↑ intra abdominal pressure

3- Sequellae of FB penetration:

- Uncommon sequels:
    - Traumatic hepatitis due to infection extended through gastroepiploic artery to liver producing liver abscess.
    - Splenic abscess
    - Diaphragmatic abscess
    - Renal abscess
    - endocarditis
- Common sequels (see diagram on the next page):


Common sequels
Acute local peritonitis
Recover
Chronic local peritonitis
Acute diffuse peritonitis
Acute pericarditis
Recover
Vagal indigestion
Diaphragmatic hernia
CHF
Death
Rupture coronary A
Death
Chronic pericarditis

Symptoms:

1- Fever

2- Complete anorexia

3- Sharp decrease in milk production

4- Animal walks, stand, and lie down with caution

5- Standing with arched back and tucked up abdomen

6- Pain during defecation and urination due to staining.

7- Slight recurrent tympany due to adhesion of the healed perforating wound of reticulum with abdominal wall.



Arched back and tucked up abdomen (traumatic reticuloperitonitis)



Typical posture of a cow with traumatic reticuloperitonitis

Diagnosis:

1- Case history

2- Clinical signs

3- Pain test:

a. Pinching of wither.

b. Percussion on sternum till reaching the reticulum (xiphoid cartilage).

c. Going up and down a slope (groaning down the hill).

d. Stick test: elevation of the animal abdomen using a stick and release suddenly.

Reaction of the animal to pain test:

- Deep respiratory movement in buffaloes

- Grunting sounds and grinding of teeth in cows

e- Turning to close left side circle - difficult

4- Metal detector:

a. it may give false positive because it may detect blunt foreign bodies without causing problems of penetration of peritoneum or reticulum.

b. It may give false negative: if the FB is copper.

5- Radiographic examination to detect the localization of FB

6- Ultrasonography: to detect fibrinous changes or abscesses in reticulum

Treatment:

1- Introduction of magnetic device to collect the FB from rumen and reticulum.

2- Preservative treatment: in pregnant animal till parturition by:

a. Immobilization.

b. Keep the animal with the fore limb raised than the hind limb.

c. Decrease food intake to decrease abdominal pressure

d. Control of infection that may enter with penetration by:

i. Sulphonamides: 2-3 gm orally for 3-4 days

ii. Oxytrtracycline: 2-3 gm dissolved in normal saline and injected IP.

iii. Penicillin-streptomycin combination dissolved in saline and injected IP.

****

ENTERITIS (ENTEROPATHY, MALABSORPTION SYNDROME)

Definition:

Inflammation of the intestinal mucosa resulting in diarrhea and sometimes dysentery, abdominal pain, varying degree of dehydration

and acid-base imbalance depending on the cause, severity and location. In many cases, gastritis is associated, leading to gastroenteritis.

Etiology:

I- predisposing factors

Factors lower the animal immunity, thus increasing the pathogenicity of the causative agent to induce enteritis:

1- The newly born calves deficient in immunoglobulins are more susceptible than adults.
2- Stress of transportation.
3- Stress of deprivation of food and water.
4- Stress of weaning.
5- Prolonged use of antibacterial agents orally may alter the intestinal microflora and permit the development of superinfection by organisms which would not normally cause disease.

II – Etiological factors:

A- Infectious factors:

- The epidemiological and clinical features of diseases of cattle in which diarrhea is a significant clinical feature are described in the following table:

Etiological agent	Age & class of animal affected	Major clinical findings
I- Bacteria		
1- Enterotoxigenic *E. Coli*	- Newborn calves (3-5 d old)	- Acute profuse watery diarrhea, dehydration and acidosis.
2- *Salmonella spp.*	- All ages, outbreak occurs, stress-induced	- Acute diarrhea, fever, dysentery, high mortality
3- *Clostridium perfringens* (type B and C)	- Young well-nourished calves.	- Severe hemorrhagic enterotoxemia, rapid death.
4- *mycobacterium paratuberculosis*	- Mature cattle, sporadic, single animal affected	- Chronic diarrhea with loss of weight, long course, no response to therapy.
5- *Proteus* and *pseudomonas Spp.*	- Calves treated for diarrhea with prolonged course of antibiotics	- Chronic to subacute diarrhea, progressive weight loss, no response to treatment.

II- Viruses		
1- Rota and corona virus.	- Newborn calves, 5-21 d old, explosive outbreak.	- Acute profuse watery diarrhea, virus can be demonstrated in feces.
2- Winter Dysentery (Coronavirus).	- Mature housed cows, explosive outbreak.	- Acute epizootic transient diarrhea and dysentery lasting for 24 h.
3- Bovine viral diarrhea (mucosal disease).	- Young cattle (8-24 month old). Sporadic, but outbreak may occur.	- Erosive gastroenteritis and Stomatitis, usually fatal.
4- Rinderpest.	- Highly contagious, occur in plague form.	- Erosive gastroenteritis and Stomatitis, high morbidity and mortality.
5- Bovine malignant catarrh.	- Usually mature cattle, sporadic but small outbreaks occur	-
III- Parasites Ostertagia Haemonchus, and Trichostrongylus, Oesophagostomum	- Young cattle in posture	- Acute or chronic diarrhea, dehydration, hypoproteinaemia, fecal examination.
IV- Protozoa		
1- Eimeria	- Calves over 3 weeks old up to 12 month of age. Outbreaks common.	- Dysentery, tenesmus, fecal examination is diagnostic.
2- Cryptosporidium spp.	- calves 5-35 days of age	- Acute diarrhea
V- Mycotic Candida spp.	- Young calves following prolonged use of antibacterials.	- Chronic diarrhea, no response to treatment.

B- Chemical factors:

Arsenic, copper, mercury, molybdenum, poisonous plants, nitrates	- all ages affected, history of access to the substance, outbreaks occurs	- all severities of diarrhea, dysentery, abdominal pain, dehydration, toxemia, nervous signs may occur

D- Nutritional deficiency.

Copper deficiency, conditioned by excess molybdenum	Usually mature cattle in pasture with high levels of molybdenum	Subacute and chronic diarrhea, osteodystrophy, no systemic effect, hair color changes.

E- Dietary:

1- Overfeeding.	- Young calves overfed on milk.	- Mild diarrhea, feces pale and yellow
2- Simple indigestion.	- Change in ration of mature cows (hay to silage) or grain to feedlot cattle.	- subacute diarrhea, normal in 24 hour

Pathogenesis:

The effect of different etiological factors is mainly localized to the small intestine, producing three sequential pathogenic pathways:

1- Inflammation or necrosis of intestinal mucosa.

2- Defect in the secretion/ absorption.

3- Osmotic disturbance.

1- Inflammation or necrosis of intestinal mucosa.

This inflammation results in a net increase in the fluid production, inflammatory products, and a reduction in the absorption of fluids and electrolytes. Examples are many of the diseases caused by bacteria, virus, fungi, protozoa and chemical agents summarized in the previous table. Many bacteria and virus multiply in the brush border cells of intestinal mucosa, thus reduced activities of brush border enzymes, decreased carrier-protein function, decreased mucosal absorptive surface area, and interfered with final transport of nutrients into the circulation.

2- Osmotic disturbance:

The osmotic effect occurs when a substance concentration in the lumen of intestine is much greater than the intestinal cells (hypertonic solution), leading to osmotic movement of excessive amount of fluid into the lumen of intestine, leading to watery feces and diarrhea. Examples are the saline purgative and the overfeeding and indigestible feeds. In addition, malabsorbed nutrients exert strong intraluminal osmotic effects that diminish intestinal and colonic absorption of water and electrolytes, resulting in diarrhea. This may be exacerbated if mucosal damage is accompanied by intestinal inflammation, which can cause secretory and permeability diarrhea.

3- Secretory –absorptive imbalance

In some cases, such as colibacillosis, the intestinal secretion is increased beyond the absorptive capacity leading to loss of fluids and diarrhea. The increased intestinal secretion is attributed to an increase in the cyclic adenosine monophosphate, wich in turn stimulated by prostaglandins.

- Therefore, in acute diarrhea, large quantities of fluid, water, and electrolytes (sodium, chloride and bicarbonate) are lost. The fluid move out of the intravascular compartment first, then out of extravascular compartment, followed lastly from the intracellular space, leading to dehydration and hyponatremia. Loss of bicarbonate leads to metabolic acidosis. With severe acute diarrhea, the circulating blood volume is reduced, resulting in diminution of perfusion of liver, kidney and peripheral tissue. This results in uremia, anaerobic oxidation and lactic acidosis, which accentuate the metabolic acidosis. Hyperventilation occurs in some animals to compensate for acidosis.
- In chronic enteritis, the intestinal mucosa becomes thickened and mucus excretion is stimulated and the intestinal reabsorption is also decreased but not to such a degree as in acute enteritis. Therefore, the feces is usually soft homogenous and containing excessive amount of mucus. However, no signs of dehydration or electrolyte loss because the animal usually drink and absorb sufficient water to maintain normal hydration.
- In parasitic enteritis, there is always hypoproteinemina with subcutaneous oedema.

Clinical findings:

Specific signs are described under specific diseases, but the general clinical finding in enteritis in cattle are:

A- Diarrhea

a. Severe watery diarrhea, sometimes dysentery, and often tenesmus in acute enteritis.

b. The feces is watery and profuse in case of lesions of small intestine.

c. Small volume and soft feces with excess quantity of mucus occur in case of lesions of large intestine.

d. The presence of blood or fibrinous cast, indicate severe inflammatory lesion of intestine.

e. The feces is voluminous, soft and odoriferous in case of dietary diarrhea.

B- Dehydration

1. Dehydration is usually marked in acute diarrhea (10-12 hr after onset of diarrhea).
2. Signs of dehydration include dry muzzle, sunken eye, shrunk skin and oliguria.
3. Dehydration can be assessed by tinting of the skin.

C- Weight loss

a. Chronic weight loss associated with chronic diarrhea may indicate John's disease.

b. Moderate weight loss, profuse diarrhea with normal hydration and depigmentation of hair and indicate copper deficiency conditioned by excess molybdenum in diet.

c. Weight loss with profuse diarrhea occurs also in intestinal heminthiasis.

D-Systemic reaction

Septicemia, toxemia and fever occur mostly in infectious enteritis, such as salmonellosis and colibacillosis

E- Peripheral circulatory collapse

Lack of perfusion of liver, kidney, and peripheral tissues due to dehydration lead to uremia and lactic acidosis.

F- Acid-base and electrolyte imbalance

Loss of bicarbonate leads to metabolic acidosis manifested by hyperventilation. Hyponatremia leads to muscle weakness.

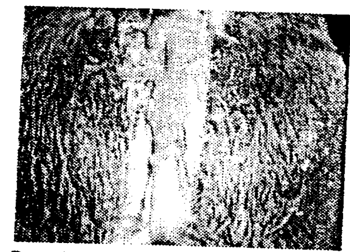
G- Abnormalities in heart rate

Tachycardia or bradycardia and cardiac arrhythmia may occur depending on the degree of acidosis and electrolyte imbalance.

F- Abdominal pain

a. Abdominal pain in calves with enteric colibacillosis is manifested as intermittent bouts of stretching and kicking at the abdomen.

b. In adults, abdominal pain is associated with salmonellosis, lead and arsenic poisoning.



Bacterial enteritis in a calf
(soiling of perineum and hindquarters)



Yellow colored feces soiling hindquarters (enteritis)



Soiling of perineum and hindquarters



Soiling of perineum (calf diarrhea)

Diagnosis

I- History

II- Clinical signs

III- Lab. Diagnosis:

a. Fecal examination: to determine the presence of causative parasite, fungi, bacteria, virus ... etc.

b. Blood analysis:

i. Haemoconcentration.

ii. Hypochloremia, hyponatremia and reduction of bicarbonate (metabolic acidosis).

iii. Hyperkalemia is possible in severe metabolic acidosis.

iv. Increased blood urea nitrogen (BUN) due to inadequate perfusion of kidney associated with dehydration leading to renal insufficiency.

Treatment:

Five goals of treatment should be fulfilled:

1- Temporary withdrawal of the diet if necessary.

2- Removal of the causative agent (Anthelmentics against parasitic enteritis antibacterial against bacterial enteritis, non specific symptomatic treatment for viral enteritis..... etc

3- Replacement of lost fluids and electrolytes.

4- Intestinal protectants and adsorbents.

5- Use of anti-diarrheal drugs e.g. drugs inhibit secretion and control intestinal hypermotility if necessary.

A) Temporary withdrawal of the diet:

If the diarrhea is dietary in origin, the feed should be removed until the animal is fully recovered..

(B) Antibacterials:

1- The use of antibacterials either orally or parenterally, or by both routes simultaneously.

2- Parenteral preparations are indicated in animals with acute diarrhea. toxemia and fever (many antibacterials when given parenterally are excreted by the liver into lumen of the intestine).

3- Oral preparations may be sufficient in cases of subacute diarrhea with minimal systemic effects.

N.B:

-Oral antibacterial preparations should not be used for more than 3 days to avoid superinfection.

-Type and doses of antibacterial commonly used in bacterial enteritis are described under each disease.

(C) Fluids and electrolytes:

To correct the 3 major abnormalities of dehydration, acidosis and electrolyte deficit.

-When severe acidosis is suspected, a 5% hypertonic soln. of bicarbonate is given IV at a rate of 5-7 ml/kg B.W at a speed of 100 ml/ minute.

-Administration of electrolyte solution in quantities necessary to correct the dehydration. :

(a) In severe dehydration (equivalent 10% of B.W), large amount of fluids are necessary at rate of 100-150 ml/kg B.W per 24 hours I/V and I gm of Kcl mast be added for each litre of fluid to overcome hypokalemia. Used successfully to correct dehydration.

(b) In animals, which are not severely dehydrated, oral route can also be used successfully to correct dehydration.

D- Intestinal protectants and adsorbants :

Kaolin and pectin mixtures are used widely to coat the intestinal mucosa, inhibit secretions and increase the bulk of faeces in horses with enteritis

E- Antidiarrheal drugs :

Antisecretory drugs for treatment of diarrhea due to hypersecretory activity. For example: atropine sulphate, chlorpromazine Hcl and prostaglandin inhibitors.

* Pathogenesis of enteritis :-

Etiological factors

- inflammation or necrosis of mm → Bact. & v. multiply in the brush border cell of Int. villi → decrease carrier Ptn function → decrease mucosal absorptive surface area → decrease transport of nutrient into circulation
- secretion absorption defect → colibacillosis → the Int. secretion is ↑ beyond the absorptive capacity → ↑ in the cyclic adenosine monophosphate → Loss of Na^+, cl, HCO_3 & water → Metabolic acidosis dehydration hyponatriemia & hypochloriemia
- osmotic disturbance → overfeeding & indigestible feeds → malabsorbed nutrients exert strong intraluminal osmotic effects → movement of excessive amount of fluid into the lumen of intestine → watery feces & diarrhea.

RESPIRATORY SYSTEM IN RUMINANTS

1- RHINITIS (CORYZA)

Definition:

Rhinitis means inflammation of nasal cavity which may be acute or chronic, unilateral or bilateral, characterized clinically by sneezing, wheezing and sterdor during inspiration and nasal discharge which may be serous, mucoid purulent or blood-tinged in consistency depending on the cause.

Etiology:

1- Infectious bovine rhinotrachitis (IBR).
2- Malignany catarrhal fever.
3- Rinder pest
4- Mucosal disease.
5- Adeno viruses 1.2 and 3
6- Syncytial vurus infections.
7- Rhinosporidiosis (caused by fungi).
8- Schistosoma nasalis (blood fluke).
9- Allergic "summer snuffles" = atopic rhinitis.

Clinical signs :

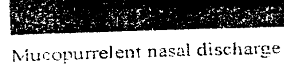
Rhinitis is of minor importance as a disease process. Its major importance is as indication of the presence of some specific infectious disease.

- The cardinal sings in rhinitis are:

1- Nasal discharge which is usually serous initially but soon becomes mucoid, and in bacterial infections, purulent.
2- Erythema, erosion or ulceration may be visible on inspection of nasal mm.
3- Sneezing in the early acute stages which followed in later stages by snorting.

4- Associated regional L.n may show signs of inflammation

N.B: Chronic, unilateral purulent nasal discharge lasting several weeks or months may suggest nasal mycosis (sporidiosis).

Mucopurrelent nasal discharge



Congestion, erosion and necrosis of the nasal and oral mucosae and the muzzle

Diagnosis:

I) History.

II) Clinical: signs.

III) Lab. Diagnosis:

Examination of nasal swabs of scabs of scraping for bacteria, inclusion bodies or fungi may aid in diagnosis.

IV) Endoscopic examination:

Endoscopic examination using a flexible fiberoptic endoscope is very useful for the visual inspection of lesions affecting the nasal mucosae which are not visible externally.

Treatment:

(A) Specific treatment to control the causative agent as described under the specific diseases.

(B) Non specific symptomatic treatment as follow:

1- Remove the thick tenacious exudates which causing nasal obstruction using warm boric acid, then irrigate the nasal cavity with physiologic saline or mixture of saline and antibiotics to provide symptomatic relief and minimize secondary bacterial rhinitis.
2- Spay-up a nasal decongestant into the nostrils to provide some relief. E.g. Delta rhinol, prosiline... etc.
3- Culture and sensitivity testing to assist the selection of suitable antibacterial agents (antibiotic or sulphonamide), which may be helpful in controlling secondary bacterial infection.

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2-LARYNGITIS, TRACHEITIS, BRONCHITIS

Definition:

Inflammation of air passages including larynx, trachea and bronchi. They are all characterized by coughing, noisy inspiration and some degree of inspiratory embarrassment.

Etiology

1. IBR (bovine herpes virus 1)
2. Calf diphtheria (necrotic laryngitis)
3. Haemophilus somnus
4. physical factors: inclement weather and dusty environment.

Pathogenesis

Irritation of mucosa causes frequent coughing and swelling that results in inspiratory dyspnea.

Clinical findings

1- Coughing: dry non-productive and easily induced by grasping the trachea or larynx. The cough becomes exudative with much mucous after secondary bacterial infection following IBR
2- Inspiratory dyspnea manifested by roaring or stridors

3- Fever and toxaemia in viral infection followed by secondary bacterial infection and affected animal cannot eat or drink.
4- In acute laryngitis, the soft tissue around the larynx is enlarged and painful.

Diagnosis:

1- Case history
2- Clinical signs
3- Endoscopic examination of upper respiratory tract

Treatment

1- most viral infection of the upper respiratory tract resolve spontaneously if the animal has rest away from the dusty food, or inclement weather.
2- Antibacterials:
   i. Sulphathiazine: IV for 3-5 days
   ii. Broad spectrum antibiotics: penicillin-streptomycin or oxytetracyclines.
3- Anti-inflammatory: dexamethasone to reduced inflammatory edema at larynx.

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3-CALF DIPHTHERIA

Definition:

It is a disease of calves affecting the oral cavity and larynx causing necrotic stomatitis or necrotic laryngitis and characterized by high fever and swelling and ulceration of the affected structures.

Etiology :

- the diseases considered necrobacillosis i.e. caused by Fusobacterium necrophorum (sphaerophorus necrophorus) preceded by traumatic injury

of mucous membranes of oral cavity or larynx example by coarse feed or feed containing an excessive quantity of thistles or tough stems as a predisposing factor.

Clinical findings:

1- Calf diphtheria usually occurs as necrotic stomatitis in calves less than 3 months of age and as necrotic laryngitis in older calves.

2- The calf with necrotic stomatitis has:

a) Difficulty in nursing.

b) Depressed appetite.

c)Temperature may rise up to 40°C.

3- The calf with necrotic laryngitis has:

a) Loud wheezing (most prominent sign in severe cases).

b) Temperature may rise up 41°C

c) Salivation and protrusion of the tongue.

d) Nasal discharge and rapid respiration.

e) Cough as the luns often become involved .

f) Dehydration and emaciation.

g) Untreated calves succumbing pneumonia and toxaemia within 2-7 days.

Lesions:

1- Necrotic ulcers on oral and pharyngeal m m.

2- Croupous or diphtheritic membranes.

Treatment:

1- Isolation of affected animals.

2- Antibiotics and sulfonamides are recommended.

(a) Antibiotics:

Antibiotics of choice are; Penicillin (procaine penicillin-G aqueous . suspension 5,000-10,000 I.U/L.b I/M).

Or- Penicillin and streptomycin (5-10 mg/Ib dihydrostreptamycin aqueous suspension I/M).

or-Chlora m phenicol (5-10 m g/Lb t.i.d.)

(b) Sulfonamides:

Sulfonamides of choice are:

- Sulfamerazine sodium 130 mg/kg b.wt. followed by 65 mg/kg daily for at least 4 days.
- Sulfamethazine sodium orally or preferably I/V i3Q mg/kg b.wt. followed by 65 mg/kg orally daily for at least 4 days.

4- PNEUMONIA

Definition:

Pneumonia means inflammation of the lung parenchymia usually accompanied by inflammation of the bronchiole (broncho-pneumonia) and often inflammation of the pleura (pleuro pneumonia).

Etiology:

(I) Predisposing factors:

Predisposing factors weaken the defense mechanisms of the ani m als e.g:

1- Inclement weather.

2- Poor ventilated housing.

3- Poor nutrition.

4- Stress of transportation. .

(II) Infectious causes:

a- Pneumonic pasteurellosis (shipping fever):

- Pasteurella haemolytica, pasteurella multacida with or without parainfluenza-3 virus.

b- Enzootic, pneumonia of calves:

1- Parainfluenza-3.

2- Adenovirusv 1,2 and 3- Rhinovirus

4- Bovine respiratory syncytial virus.

5- Reovirus.

6- Bovine herpes virus 1 (IBR).

Plus:

1- Chamydia sp.

2- Mycoplasma sp.

3- Pasteurella sp.

4- Actinomyces (corynebacterium) pyogenes.

5- Streptococcus sp.

6- Bedsonia sp.

7- Actinobacillus actinoides.

c- Viral interstitial pneumonia:

In recently weaned beef calves caused by bovine respiratory syncytial virus.

d- Contagious bovine pleuropneumonia:

Mycoplasma mycoides.

e- Verminous pneumonia:

1- Dictyocaulus viviparus.

2- Massive infestation with ascarid larvae.

f- Pneumonic tuberculosis:

sporadically, and caused by, mycobacterium bovis.

g- Klebsiella pneumoniae: In calves.

h- Fusobacterium necrophorus pneumonia:

Sporadically and accompanied calf diphtheria.

Clinical findings:

1- Rapid. shallow respiration, is the cardinal sign of early pneumonia.

Dyspne occurring in the later stages when much of lung tissue is non-functional.

2- Cough: which is:

- Dry, frequent, hacking cough in interstitial pneumonia.
- Moist., painful cough in bronchopneumia.
- Cyanosis: Not a common sign, occurs only when large areas of the lung are affected.

4- Nasal discharge: may or may not present depending upon the amount of exudate present in bronchioles and whether or not there is accompanying inflammation of the upper respiratory tract.

5- Abnormal odour of the breath:

- Decay when there is a large accumulation of inspissated pus
- Putrid when lung gangrene is present.

6- Auscultation of the lungs:

- In the early congestive stage of broncho-pneumonia and interstitial pneumonia there is increased tronchial sound (breah sound).

-crackles (moist rales) develop in broncho-pneumonia as bronchiolar exudatation increases.

- Clear, harsh bronchial sounds are audible in uncomplicated interstitial pneumonia.
- Loud bronchial, sound when complete consolidation in either form occurs (consolidation also causes increased audibility of heart sounds).
- Pleuritic friction rub in early stages when pleurisy is also present, and muffling of bronchial Sounds in the late exudative stages.

7- In acute bacterial bronchopneumonia, there is toxemia, fever anorexia, depression and tachycardia.



Coughing with extention of the head and neck (parasitic pneumonia)



Extension of the head and neck, nasal discharge, and coughing (Pneumonic pasteurellosis).



Dyspnea in late stage of pneumonia (cow)

Diagnosis:

I) History II) Clinical signs III) Endoscopic examination

IV) Nasal swabs.

Treatment

1- Isolation of the affected animals especially if infectious, disease suspected, isolation must be in warm, well ventilated, draft free place and provide with ample fresh water and light nourishing food (parenteral feeding if animal not eat).

2- The choice of antibacterial, agent (antibiotic or sulfonamide, based on culture and sensitivity testing.

or

the choice of antiparasitic agent if verminous pneumonia is suspected.

3- The use of corticosteroid as an antiinflammatory agent e.g. Dexamethazone.

4- The use of antihistaminic e.g. avil

5- The use of bronchodilator to i m prove ventilation e.g aminophylline and theophylline.

6- The use of expectorants according to the type of cough:

- When cough is painful and exhausting and the secretion tenacious, sedative expectorant such as ammonium or potassium salt. stimulate secretion of protective mucous and lessen coughing.

- When,cough is soft and bronchial exudate voluminous as in chronic bronchopneumonia stimulant expectorant is more valuable. .

- When cough is exhausting and interferes with activity but there is little exudation an anodyne expectorant, such as belladonna, codine, morphine or heroin is indicated.

URINARY SYSTEM DISEASS IN RUMINANTS

Clinical manifestations of urinary system diseases in ruminants

I) DYSURIA AND STRANGURIA

Dysuria: Is difficult or painful urination.

Stranguria: Is slow and painful urination.

Causes: May be either:

a) Inflammation of the urethra, U.B. or both.

b) uretheral obstruction.

c) Neurological conditions that prevent normal emptying of U.B. (e.g, paralytic bladder).

Table of primary causes of dysuria or stranguria in ruminants

Cattle and buffaloe	Sheep and goats*
1- Urethral calculi	1- Urethral calculi
2- Hemorrhage into urinary tract	2- Cystitis
3- Urethral swelling secondary to calving.	3- Injury to urethera or swelling around urethera.
4- Penile or preputial injury.	4- Spinal cord trauma or pressure.
5- Cystitis.	
6- Rabies	
7- Pelvic bladder.	
8- Sacral fracture	
9- Extradural lymphosarcom a in the vertebral canal.	
10-Spinal cord trauma or pressure.	

4- Normal urination in ruminants is passive, and straining or ,groaning associated with urination should not be observed (horses, voided urine very activley and forcefully and normally may groan and strain on urination).
5- Dysuria /or stranquria in ruminants associated with urine "dribbling" in male sheep and goats with calculi.
6- Vocalization is associated with dysuria in male goats with uretheral ruction.
7- Urine scalding of perineal region or rear legs may be noted in ruminants with dysuria and/or strong uria.
8- Intermittent prolapse of the prepuce may be another clinical signs in males suffering from dysuria and/or stranguria.

II) HAEMATURIA

Haematuria means blood in the urine, it may appears as gross blood clots passed at the beginning, during, or at the end of urination or as more uniform red discoloration throughout urination without clotts.

Organ	Condition
a- Kidney :	1} Pyelonephritis 2} Trauma 3} infarction 4} Malignant catarrhal fever..
b- urinary bladder:	1- Calculi 2- Cystitis. 3- Papilloma. 4- Bracken fern or other bleeding diathesis.
c- Urethra:	1- Calculi 2- Trauma

3- Urethritis

Table of causes of haematuria in ruminants.

III) PYURIA

1- pyuria means purulent debris in the urine.

2- This may be either microscopic or gross observation.

3- Dysuria, stranguria, crystalluria, urine scalding of the perineum, or even haematouria may accompany the pyuria.

4- A fever may or may not be present, but if the pyuria is originating from the upper urinary tract, the infected animal often is systemically ill.

5- Pyuria may be result of an inflammatory and septic disease process.

6- if the sepsis is caused by a bacterial infection, bacteriuria will be present.

IV) UREMIA

1- Uremia means; the presence of excessive urinary constituent in the blood and their systemic toxic effect.

2- Clinical signs of uremia seen in large animals are:

a) Depression, and anorexia.

b) Seizures and/or encephalopathy may occur in some cases with severe uremic episodes.

c) Weight loss, oral lesions, gastrointestinal ulcers, meiena, and accumulation of excessive dental tarter.

3- uremia may be the result of either:
a) Acute ranal failure

Or b) Chronic renal failure.

Table of causes of chronic renal failure in ruminants

Glomerular causes	**Tubulointerstitial causes**
1- Glomerulonephritis (rare)	1- Chronic pyelonephritis
2- Renal amyloidosis	2- Chronic obstruction
	3- Any of the vascular, toxic, or septic causes of acute renal failure.

Table of acute renal failure in ruminats

Septic causes	Hemodynamic causes	Mycotoxin	Endogenous causes	Drugs
1- Renal necrosis 2- Pyelonephritis 3- Septic mastitis. 4- Septic metritis.	1- Renal vein thrombosis. 2- Severe bloat 3- Shock 4- Heart failure	1- Ochratoxin. 2- Citrinin	1- Hemoglobin. 2- Myoglobin	1- Sulfonamide 2- Amino- glycoside antibiotics. 3- Oxytetra cycline. 4- Monensin.

N.B: Toxic plants (oak, a corn, rumex. Etc.) and some chemicals (arsenic, mercury, sodium fluoride, chlorinated hydrocarbons… etc.) are of the recorded cases of acute renal failure in ruminants

v) POLYURIA

1- Polyuria means the passage of abnormally large amounts of urine.

2- Palyuria may be caused by:

a) Normal response to excessive fluid and/or electrolytes intake,

b) Renal failure (acute and chronic).

c) Diabetes mellitus (rare)

d) Hyperglycaemia,

e) Diabetes insipidus (central, nephragenic),

f) Salt disturbances deficiency or toxicity).

g) Steroid administration (Cushing's disease "rare"')

h) Diuretic

k! Severe deficiencies of chloride, potassium, or urea.

VI) URACHAL LEAKAGE OF URINE

Incomplete urachal closure in newborn ruminants is visible by dripping of urine from the ventral abdomen and/or swelling of the umbilical area,

VII) CRYSTALLTLRIA

- Crystalluria means the presence of crystal in the urine.
- Crystalluria lead to stone formation.

VIII) MICTURITION

- Hind legs of cows are separated during normal micture, or may not do so. Contracting the abdominal wall, exerting pressure on the U.B. evacuated it even during walking.
- Male cattle urination is a dribbling process, urine being passed frequently in small amounts which dribble away from the prepuce.

IX) FREQUENCY

1- Increase frequency may be due to increased volume of urine e.g., increased fluid intake, the action of diuretics and cold weather.

==

2- increased frequency of urination may arise from urethral irritation due to cystitis.

- Vesical calculi by irritating the m m of L:B. stimulate contraction and this initiate urination.

- Infrequent urination may be due to reduction in the amount of urine, which will occur if.- there is dehydration from any cause, or a sudden in blood pressure.

X) INCONTINENCE (Inability to retain urine)

- Occurs as a result of paralysis or weakening of the sphinctor of U . B., or as a result of diminished sensitivity of the urethral mm.



X I) Oliguria or anuria.

X II) Abnormal urine colour.

X III) Transparency and turbidity.

X I V) A bnormal odour, of urine. (in practice)

X V) Abnormal Sp. G. of urine.

X VI) Abnormal P H of urine.

X VII) Abnormal constituents of urine.

XVIII) Abnormal urine deposites.

URINARY SYSTEM DISEASES

1- Renal ischemia:

2- Renal amyloidosis.

3- Pyelonephritis.

4- Embolic suppurative nephritis.

5- Glomerulonephritis.

6- Acute tubular necrosis.

7- Interstitial nephritis.

8- Caprine protozoal parasites of kidney.

9- Urinary calculi.

10-Cystitis.

11-Bovine enzootic haematuria.

1-RENAL ISCHEMIA

Definition:

Renal ischemia is a pathological condition attributed to acute or chronic reduction in blood flow through the kidney and characterized by transitory oliguria followed by anuria and uremia.

Etiology:

Reduction in blood flow is usually the result of circulatory failure:

A- Acute renal ischemia:

Usually caused by general circulatory emergencies, e.g: shock, dehydration, acute haemorrhagic anemia and acute art failure.

B- Chronic renal ischemia:

Usually caused by chronic circulatory insufficiency e.g: congestive heart failure.

Pathogenesis:

A- Reduction in blood flow result in an immediate reduction in glomerular filtration and subsequently the following:

1- Elevation in the levels of normally excreted metabolites in blood stream. e.g: BUN i.e. prerenal uremia.

2- Concomitant reduction in urine flow i.e. oliaguria or even anuria.

B-Reduction in blood flow if severe and persists for long period the resulted reduction in blood flow usually followed by : Anoxic degenerative lesions in the renal parenchyma (nephrosis) vary from tubular necrosis to cortical necrosis.

Clinical findings:

1- Renal ischemia does not usually appear as a disease entity because it is masked by the clinical signs of the primary disease.

2- Oliguria or even anuria, associated with the clinical picture of uremia.

3-The only observed clinical signs of renal ischemia may be the clinical picture of uremia associated with oliguria or anuria.

Diagnosis:

A- History

B- Clinical findings

C- Lab. diagnosis:

1 - Determination of urea nitrogen in the blood (BUN) usually elevated.

2- Proteinuria, particularlly in the irreversible damage stage to the renal parenchyma (especially glomerular cortical necrosis).

N.B. : Evidence of oligura + azotemia in the presence of cirulatory failure suggests renal ischemia.

Treatment:

1- Correction of the circulatory disturbance at the earliest opportunity

2- Supportive treatment if renal damage has occurred (as suggested for the treatment of renal failure) usually fluid therapy.

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✓PYELONEPHRITIS
(CONTAGIOUS BOVINE PYELONEPHRITIS)
OR (ENZOOTIC BOVINE PYELONEPHRITIS)

Definition:

- Pyelonephritis is a subacute or chronic, purulent inflam illation of the kidneys and pelvic portion of the kidneys.

- C.B. pyelonephritis or E.B. pyelonephritis is a highly fatal specific infection of the urinary tract of cattle caused by corynebacterium renaie

and characterized by chronic purulent inflammation of the kidneys, ureters and bladder.

- The disease affect adults only and in sporadic cases, but may be a series suggesting origin in one bull and relationship to mating events.

Etiology:

1- The specific cause of C.B.P. is corynebacterium renale (possibly 4 serotypes, type 1 appears to be most pathogenic, and all 4 types are capable of stimulating production of complement-fixing antibodies which give cross-recations with mycobacterium johnei)

2- Corynebacterium pyogenes, carynebacterium pseudatuberculosis coryne equli, E. coli and Staph. aureus and Streptococci and unidentified diphthroid bacilli are sometimes found in urinary tract of cattle affected with pyelonepheritis either alone or associated with cor. renale (mixed infection).

N.B.:

The disease Ist reported in U.S. (Boyd 198I),:

Epidemiology:

1- Clinical cases of disease appear sporadically.

2- The disease highly fatal, unless the appropriate treatment instituted early.

3- The disease of economic importance in cattle - economic losses from the disease due to deaths of the affected animals.

4- The disease essentially a bovine disease, but sheep are occasionally affected.

5- Cows much more susceptible than bulls and cow before maturity (unbred heifers) are seldom affected (not infrequent).

6- Increase clinical cases incidence of the disease in colder season of year i.e. winter (relation of cold to kidney diseases is found in nephritis in

man).Ibecause of the persistence of pyelonephritis as an endemic affection in high-producing (heavily fed) dairy herd, it has been suggested that a high protein diet may increase susceptibility to the disease (predispose to an attack).

N.B:

C.B.P. is regarded therefore one of the stress-related diseases (stress of cold or uncomfortable weather; stress of high production and advanced. pregnancy and stress of heavy feeding).

7- The short wide and often traumatized urethra of cows, probably offers a predisposition to infection by allowing the enterance of the organism into the U.B.

8-C.renale has been cultured from the vulva, vagina vestibule and penile sheath prepuce and urethra of bulls) of apparently normal cattle. (The bacteria that have been isolated produced pyelonephritis in lab. albino mice by I.V route).

9- The vulva is thought to be the portal of entry in the cow. This view supported by the very firm adhesion of C. renale to epithelium cells of bovine vulva and typical lesions can be established experimentally in some animals by the introduction of the organism into U.B. (Wester 1927) producing the disease experimentally by introduction of sterile sand and pure culture of C. renale into U.B. followed by massage).

10 - Evidence reveled that, the disease transmitted by contact, as grooming animals with contaminated brushes vulvar contact with urine-soiled bedding, tail switching and the use of non-sterilized catheters or obstetric instruments can induce the disease.

11- Venereal transmission seems to be a likely means of spread in animals bred by natural service (venereally spreed). This supported by cessation of cases when A.I. is used.

12- Clinically affected or clinically normal "carrier" cows are probably the principal source of infection.

13- C.B.P. may develop secondary to bacterial infection of lower urinary tract.

N. B:

Spread of pyelonepheritis from "embolic nepheritis" of haematological origin such as pseudomans aeruginosa septicemia in cattle.

Pathogenesis:

1- Infection of urinary tract should be present.

2 - Stagnation of urine which permit the multiplication must be present.

3- Infection progress up the urinary tract.

4- Stagnation of urine may occur as a result of:

a) Blockage of ureters by the inflammatory swelling or depris

or by b) Obstructive urolithiasis. or by c) Pressure from - uterus in pregnant cows.

5- infection ascends the ureters not always bilaterally and invades the renal pelvis.

6- Infection extended to the renal medulla and then may extend to renal cortex.

7- Toxaemia and fever may result if the renal involvement is bilateral obstruction of urinary outflow, occurs and death follows.

Clinical findings:

1- The first sign may be passage of smoky or blood stained urine.

2- In some other cases the first sign may be an attack of acute colic, such attacks due to the obstruction of the ureter, and renal pelvis by pus or tissue debris.

3- Fluctuating temperature, depraved appetite, loss of condition, decrease in milk yield over a period of weeks,

4- Sometimes systematic reaction.

5- Frequent painful urination.

6- Voiding smooky or tubid urine due to presence of pus, blood, mu... tissue debris in the urine.

7- Pain on pressure at. the transverse processes of the right lumber region.

8- Rectal palpation reveal:

a) Thick tender wall of U.B.

b) Cord-like one or both ureters.

9- One or both kidneys show enlargement and absence of lobulation and pain on palpation.

Diagnosis:

A) History. B) Clinical signs

C) Lab. diagnosis

1- Albumin, R.B.Cs, W.B.Cs, epithelium cells and casts are present in urine.

2- Corynebacterium renale isolation from suspected urine.

Treatment:

1- Large massive dose of penicillin for long duration is the drug choice 7-8 million I.U for 7-10 days at least.

2- Acidification of urine by administ. Of monobasic sodium acid phosphate 125 gm daily for 7 days at least.

3- Urinary antiseptic e.g: 15 gm hexamine 40% solution I/V given after acidification of urine.

4- Easily digestible nutritious food, fresh water, barley water ,barley and tonics as arycyl.

✔EMBOLIC SUPPURATIVE NEPHRITIS (RENAL ABSCESS)

a-Lodging of emboli of bacteria in the renal issue after any bacterimia or septicemia.

b- The origin of the emboli may be sporadic cases such as:

1- Suppurative lesions -in uterus, udder, navel, and peritoneal cavity in cattle.

2- Valvular endocarditis.

Clinical findings:

1- Usually there is insufficient renal damage to cause signs of renal dysfunction, but signs of toxaemia and the primary disease are usually present.

2- Enlargement of the kidney may be palpable on rectal examination.

3-Spread of suppurative emboli to the renal pelvis may cause a syndrome very similar to that of pyelonephritis.

Diagnosis:

a) History

b) Clinical signs

c) Lab. diagnosis:

1- Proteinuria.

2- Microscopic examination of urine reveal Pus and blood cells.

3- Culture of urine at the time where proteinuria Occurs may reveal the bacteria infecting the embolus.

N.B:

Differention of embolic suppurative nepheritis from pyelonephritis is difficult unless the later is accompanied by detectable cystitis or urethritis this especially because enlargement of the kidney occurs in both conditions and urine analysis are the same when embolic nephritis invades the renal pelvis in the cows.

Treatment:

1- Long duration (7-10 days), massive dose of antibiotics or sulphonamide if the causative bacteria can be isolated and sensitivity determined.

2- Parenteral administration (enzymes during the same time).

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RENAL AMYLOIDDSES
(RENAL AMYLOIDOSIS AND NEPHROTIC SYNDROME)

Definition and etiology:

- Renal amyloidoses is a disease complex resulting in deposition of twisted- B-pleated sheet fibrils farmed from various proteins .
(amyloid) in the kidney.
- Nephrotic syndrome is a renal problem may develop because of extensive renal glomerular "amyloid deposite".

Pathophysiology:

1- Amyloid protein Are classified either as A A (the protein found in systemic amydoidosis acquired secondary to chronic inflammatory disease AL (the protein derived from immunoglobulin tight chains).

2- Most amyloid found in cattle is of the AA or reactive type, but an inflammatory focus is not always obvious, even at necropsy.

3- The B-pleated sheet configuration of the fibril makes amyloid resistant to solution in pbysiologic solvents and to normal proteolytic digestion.

4- The accumulation of the inert fibrils in the tissues results:

a) Pressure atrophy b) Functional disruption.

5- Glomerular deposition of amyloid results:

a) Impairment of glomerular permeability

b) Consequent loss of protein in urine.

6- Loss of low molecular weight anticoagulants such as antithrombin-III, may be responsible for renal vein thrombosis that occasionally occurs:

7- Intestinal deposit result:

a- Malabsorption b- Diarrhea.

b) Oedema formation further contributing to the diarrhea, degree of oedema in the tisssues -is proportional to the severity of the hypoalbuminemia.

9- Urermia may result: (a) Oral lesion and (b) Unhealthy gastrointestinal mucosa, as a result many uremic patients have chronic diarrhea.

10-hypoalbuminemia.

Clinical findings:

- Sporadic occurrence in cattle more than 4 years old and affected animals have: (a) Chronic intractable diarrhea (b) Weight loss. (c) Ventral oedema (d) Gross enlargement of the kidneys can sometimes be detected on rectal examination although the kidneys retain their normal shape and lobulation.

Treatment:

1- Prognosis in renal a myloidosis-affected cattle is poor and salvage is usually recommended.

2-Human cases of amyloidosis have shown clinical improvement following treatment with dim ethyl sulfoxide, but similar „ attempts to treat amyloidosis in "dogs" were unsuccessful.

✓GLOMERULONEPNRITIS

1- Golmerulonephritis is an "immunologic disorder".

2- Glomerulonephritis result either in:

a) Deposition of antigen-antibody complex in the glomerular basement membrane

b) Attachment of anti-GBM antibody to the capillary walls, with consequent impairment of glomerular perm selectivity. This leads to reduced filteration capacity and enhanced permeability to plasma proteins.

5- Glomerulonephritis is an uncommon clinical renal disease in aged sheep and goats lover 7 years. The three types that are seen are:

a) proliferative glomerulonephritis.

b) Glomerulonephritis of pregnancy toxaemia.

c) mesangiocapillary glomerulonephritis.

4- Glomerulonephritis occurs occasionally as a clinical entity in cattle. but the cattle are usually not examined until the disease process is advanced.

- Recent study failed to detect any evidence of glomerulonephritis in cattle, one report of the disease was reported in cattle persistently infected with BVD, whereas another described a mild proliferative glomeruionephritis in streers at slaughter.

The clinical signs are:

a- Weight loss

b- Chronic-diarrhea

c- Generalized oedema.

Clinical laboratory finding include:

a- Proteinuria, b- Hypoalbuminemia c)Anemia

d- Elevated serum creatinine and BUN in advanced cases.

e- Renal biopsy confirms . the diagnosis

5- Spontaneous proliferative glomerulonephritis in sheep is usually asymptonlatic. this may be of the early slaughtering age. Proteinuria recorded 30% of cases, whereas mild elevation in B U N reported in 50% in cases.

- The disease characterized by proliferation of mesangial and endothelial cells of the glomerulus sometimes accompanied by basement membrane thickeing. The disease may be im mune mediated, since IgG and complement deposition on the glomerular capillary walls are seen. The process appears not to be autoimmune because the antibodies are not directed against glomerular antigens. Some suggestion has been made not the antibodies are directed against vibrio fetus.

6- Pregnancy is a disease of m ultisystemic disturbances., A common finding in affected ewes is proteinuria and azotemia.

The renal lesion associated with pregnancy toxaemia is inflammation of all-glomeruli.

7- Mesangiocapillary glomerulonephritis, characterized by spontaneous, rapidly progressing glomerulonephritis of young lambs.

Clinical signs, include: (a) circling (b) seizure, (c) nystagmus, (d) visual deficits (e) muscle tremors (f) facial spasms (g) tachycardia k) renal erilargement and (1) pain.

Clinical pathologic changes appear before clinical signs and include: (a proteinuria (b) hypoalbuminemia (d Ureamia (d) Hypocalemia and (e) Hyperphosphatemia.

Microscopically, the lesion is a diffuse, severe mesangiocapillary glomerulonephritis.

8- Treatment of glomerulonephritis: cattle has not been described and is probably not indicated in most cases because, of the likelihood of the lesions being advanced at the time of examination.

If treatment were attempted, it would consists of "plasma" transfusions for those cows that are severly hypoproteinemic.

The use of anabolic steroids and moderate_protein and low phosphorus diet may result in a slower progression of the disease.

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ACUTE TUBULAR NECROSIS (ATN)

(Tubular nephrosis)

Etiology:

ATN is caused by:

(a) Real ischemiawhich may oocur with; 1- acute severe mastitis.

2- septic metritis, 3- abomasal torsion

4- Salmonellosis 5- pregnancy toxemia

6- the severe seplitic process

b) Nephrotaxins which may occur with nephrotoxic agents include:

1- Endogenous –nephratoxic agent e.g: bile, Hb. and myoglobin.

2- Antibacteral nephrotoxic agent e.g. tetracyclines (in septic cattle) that are given high dose for 3 or more days, sulfonamidesand aminoglyco,,ide,, (most frequent in dehydrated cattle).

3- Heavy metal nephrotoxic agents e.g. arsemsenic, mercury, cadmium and lead.

4- Plants nephrotoxic agents e.g. Amaranthus spp., Oaks, Isotropis, spp and Rumex s,pp. and Rumex spp

5- Misullaneus nephrotoxis. agents e,g. Ethylene glycol, Chorinated hydrocarbons, oxalate. mycotoxin and Monensin .

N.B.

When early signs of tubular damage are found withdrawal of the drug usually result in prompt healing-of the tubule.

Pathophysiology:

1- The renal tubules have a high metabolic rate, and thus are very susceptible V., toxins that may inactivates the cellular enzymes systems.

2- if tubular damage is widespread, the result is renal failure

3- In case of ischemic nephrosis destruction of the tubular basement membrane are making epithelial regeneration impossible and rendering the prognosis, much less hopeful .

4- Initial oliguria is often followed by diuresis because of inadequate tubular resorption, this result in loss of electrolytes and water

Clinical signs :

Of A T N are non-specific and in some cases a preexisting disorder may mask the renal problem.

- In one series of acute A T N clinical signs may be:

a- Marked depression b- Anorexia c.- Bleeding diathesis

d- Recumbency e- Polyuria g- Dehydration.

Diagnosis

I) History II) signs III) Lab diagnosis

1- Elevated serum UN and creatinine.

2- Hypocalcemia, hyperphosphatemia, hypermagnesemia, hyponatermia, hypochloremia, hypochloremia, and hypokalemia may be present.

N. B.

Cattle With peracute nephrosis may have metabolic acidosis and hyperkalemia.

(B) Urinalysis

In Early cases the following may be found:

1- Urine sp.G less than 1.022.

2-Prognosis:

3- Large number or granular casts. .

Prognosis:

-Prognosis in cattle

1- Good with early diagnosis and intensive therapy in cases of drug and plant nephrotoxins.

2-Guarded with A T N secondary to sepsis.

Treatment:

a- The objective of therapy is sustain life, until tubular epithelium regeneration occur This is manifested by:

1- Increased urine Sp.G.

2- Reduction in serum uria and creatinine.

b- Therefore treatment is aimed at (I) removal of the animal, from the source of toxins, (2) Restoring blood volume With fluids containing isotonic amounts of "calcium, sodium and chloride".

c- Mannitol (0.25 g/kg) or dopamine (3-5 ug/kg/minute) given I/V may be use in oliguric or anuric cases.

- Furosemide (1 mg/kg I/V) may be given during this infusion) and repeated every hour until urine flow is observed.

Prevention:

1- Preventing access to known nephrotoxins.

2- Maintaining adequate hydration and frequent monitoring renal parameters in animals receiving potentially nephrotoxic drugs.

INTERSTITIAL NEPHRITIS

1- Diffuse interstitial nephritis has been recorded as a cause of death in cattle, and may be in some cases be a consequence of acute tubular - necrosis.

2- Focal interstitial nephritis occurs much more commonly as an incidental finding at necropsy or meat inspection.

a- Interstitial focal nephritis in lamps has been associate with leptospira Pomona infection.

b- Interstitial nephritis in calves has been attributed to systemic coliform infections.

BOVINE ENZOOTIC HEMATURIA (BEH)

1- Enzootic hematuria occurs: cattle older than 4 Years of age.

2- BEH is a disease of uncertain etiology.

3- BEH occurs in specific areas usually where there is exposure to bracken fern (Presidium equilinum).

4- The clinical disease results from hemorrhage associated with tumors of the bladder:

-The bladder tumor have a wart like appearance and may be a single or multiple.

- The growths vary in type and may be classified histopathology as papilloma, adenoma, adenocarcinoma, transitional cellcarcinoma, sequameous cell carcinoma, fibroma, hemangima and hemangiosarcoma.

5- The main clinical signs is intermittent hematuria that has an insidious onset:

- The periods between bouts of hematuria vary from a few weeks to a few months.

-The course of the disease is usually several years, and unless the animal is culled, death eventually result from hemorrhagic anemia.

6- prognosis poor in cases of long-term BE H. Therefore, once diagnosis has been made, it is best to dispose the animal.

✓CYSTITTS

Definition:

Cystitis means inflammation of mucous membrane of U.B and clinically characterized by frequent painful urination.

Etiology:

(A) Bacterial infection:

1- Introduction of bacterial infection into the urinarry bladder after:

(a) Trauma to U.B. e.g: Vesical calculus.

(b) Stagnation of urine e.g.:Late pregnancy, difficult parturition, paralytic bladder.........etc.

2- Introduction of bacterial infection may be via contaminated catheterization.

3- Bacterial Infection (population) causing cystitis usually mixed but predominantly E.coli.

(B) Accompaniment of a specific disease:

C.B pyelonephritis in cattle which caused specifically by corynebacterium renal.

Pathogenesis:

1- Bacterial infection, frequently gain entrance to the U.B. but are removed before they invade the mucosa of the physical emptying of the urine.

2-Injury to mucosa facilitates bacterial invasion of the U.B wall.

3- Stagnation of urine is a predisposing cause for invasion of U.B mucosa by rapid multiplication.

Clinical signs:

1- Painful sensation and desire for urination due to the urethritis which usually accompanies cystitis.

2- Frequent painful urination and sometimes accompanied by granting.

3- The volume of urine passed on each occasion is usually small.

4- Animal remain in the posture adopted for urination for some minutes after the urine flow has ceased.

5- Moderate abdominal pain and moderate febrile reaction very acute cases,

6- Acute retention of urine may developed if urethra becomes blocked with blood or pus but this is unusual.

7- Chronic cases of cystitis show the same syndrome but the followings are presents:

a) The abnormalities are less marked.

(b) Frequent urination and small volume urine are the characteristic signs.

(c) Rectal examination revealed palpable inflammatory thickening of U.B wall.

N.B.

- In acute cases no palpable abnormality may be detected but pain may be evidenced.

Diagnosis:

I) History II) Clinical signs

III) Lab. diagnosis:

1- In acute cases the urine odour strong urinepherous, as well as the urine turbid due to the presence of blood and pus.

2- In chronic cases there may be no abnormality on gross inspection.

3- Microscopic examination of urine sediment or centrifuged deposits:

a) Erythrocytes.

b) Leukocytes (pus).

c) Desquamated epith. cells.

===

4- Bacteriological examination of a septic urine sediment or centrifuged deposits:

- Culture and sensitivity testing to achieve the followings:

a) Isolation of the causative agent to confirm diagnosis

b) Determination of drug or antibiotic of choice for treatment.

Treatment:

a- Parenteral treatment:

Parenteral antibiotic treatment for at least 7-14 days offer the best method for controlling the infection.

N.B:

Bladder irrigation now has been largely discarded as a method of treatment.

b- Bacteriostatic agents:

Sulphonamides, and drugs such as hexamin and mandelic acid which alter the pH of the urine, are at best bacteriostatic but their use is often followed by relapse.

c- Diuresis:

Free access to water to ensure a free flow of urine .

N.B.

Prognosis in chronic cases of cystitis is pout because of the followings:

- Difficulty of completely eradicating the infection.
- Common involvement of the kidney.

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✓RUMINANT URINARY CALCULI
(RUMINANT UROLITHIASIS, RUMINANT UROLITI#S)
(RUMINANT UROLITHIASIS SYNDROME)

Definition:

1- Urolithiasis in ruminants occur sporadically cattle buffaloe,sheep and goats, but of particular importance in "feedlots"

2- uroliths become clinically important when obstruction of urinary tract occurs.

3- Obstructive urolithiasis is almost exclusively disease of males (steers, rams, bucks, and wethers).

4- There are 3 common clinical entities occur in the urolithiais syndrome in ruminant.

a- Urethral obstruction.

b- Rupture of the urethra.

c- Rupture of the U . B.

N.B.

Nephrolithiasis, rarely induce rupture of a kidney in ruminants but sometimes causing "ureteral obstruction" in cattle (cows and bulls), leading to hydronephrosis and Pyelonephritis.

Etiology:

1-A number of theories have been advance to explain the. pathophysiology of urinary calculi formation.

2- Concentration of calculogenic crystalloid in urine to the point at which the solution become unstable and preceipitation occurs as appears in most of these theories,

3- The first step in the development of a calculus is the formation of a "crystal nidus", further growth of the nidus depends on: (a) Its ability

to remain in the urinary tract (b) Continued supersaturation of the urine with crystailoids.

N.B.

The degree of urine supersaturation depends on 4 factors: Diet, water intake, urine pH. and presence of crystallization inhibitors.

(A) Diet:

a- Silicate calculi formation:

1- Diets containing high levels of silica e.g some grasses high as 60% cause silicate urolithiasis.

2- Cattle raised on such silica-rich diets, the reticulorumen fluid contains "silicon dioxide", which is convented to "silicis acid".

3- Silicic is absorbed from GII and excreted in urine until concentration rises to values of supersaturation (especially when the water intake is low).

4- High concentrations of silicic acid from large molecular weight micelles of silica (Si-0-Si) via siloxane bonds.

5- Silica micelles coalesce and precipitate with "urine protion" to form Silicate nidus; which continue to grow in the presence or continues supersaturation of the urine.

6- Silicate calculi contain 3-4 mucoprotein-level protein are enhanced by:

- Feeding high concentrate diets.
- Inadequate dietary level of vitamin-A

b- Struvite calculi formation: (magnesium ammonium phosphates)

1- Struvite calculi mainly affect ruminants in feedlots,this especially because, they raised mainly on cereal-based rations.

2- Cereal-based ration are:

- High in phosphorous.

- Increasing the cereal to roughage ratio of the diet.

3- Increased dietary "phosphorus" intake and concentration of urine phosphates to the extent that crystallization occur.

N.B.

In one study diets high in phosphate (0.8%) resulted in calculi in 75% of the animals examined, whereas diets containing 0.3% phosphate prevent struvite calculi.

- Struvite calculi also contain 3-4% mucoprotein.
- Increased dietary "magnesium" intake has recently been experimentally to causes uroliths in growing calves. Addition of calcium but not phosphorus, to the diet appeared to protect against urolithiasis caused by high dietary magnesium.

(B) Water intake:

1- Reduced water intake is thought to result in increase concentration of "crystalloids" in urine.

2- Percent of dietary salt ingested influences water intake.

(C) Urine-PH:

1- Siliceous calculi is unaffected by change in PH "over" the normal range. Moreover, acidification of urine by addition of ammonium chloride did not reduced the concentration of silicic acid-in the urine, nor did it reduce the weight of calculi produced on diet-rich silica.

2- Sturvite calculi reduced by acidification of urine by favor dissolution of struvite cation of urine.

Epidemiology:

1- In ruminants the predominant types of Calculus art' composed of:

a) Struvite (magnesium ammonium phosphate).

b) Silicates.

c) Carbonates

d) oxalates.

2- In ruminant the formation of calculi depends on the supersaturation of wine with "calcuogenic crystalloids"

3- In ruminants the cereal-based feedlot ration are high is phosphorus and predispose to the formation of struvite caclui.

4- In ruminants, increasing the concentrate to roughage increases the amount of sediment in the urine, therefore, increasing the calcium content of the diet "protects" against formation of calculi reducing the amount of phosphorus absorbed.

5- In Sheep , pelletting of a sheep ration increased both urinary phosphorus concentration and urinary calculus formation (although the mechanism is not understood).

6- Water intake has a significant effect in the degree of saturation of the urine, therefore reduced intake of water as in winter is thought to be important factor in the increased prevalence to be an that time.

7- In ruminant, castrated males are most susceptible to "obstructive urolithiasis because the bore of the urethera significantly smaller in astrates than in bulls".

8- In ruminants, the females are less susceptible to urolithiasis due to the short and wide urethra which ensure passage of any formed calculi v- the rapid flow of urine at micturition.

9- Calculus is usually present at the sigmoid of males.

Clinical signs:

A) Urethral obstruction:

1- Urethral pain manifested by strains in an attempt to urinate (sometimes) to the extent that rectal ,prolapse occurs), restlessness, kicking at the belly and switching the tail in steers.

2- Dribbling of blood stained urine particularly if the urethral obstruction is incomplete. But if obstruction is complete is complete anuria) the preputial hairs are dry and calculi may be deposited on them.



Mineral deposits at the preputial hair of steer

3- Pulsation of the urethra at the ischial arch often present and (an be palpated rectally.

4- Swelling at "the sigmoid flexure" resulting from calculus lodgement" may be detected, and hypersensitivity to palpation in this area is significant.

N.B:

In sheep and goats pulse and respiratory rate frequently elevated,a nd rams, bucks and wethers calculus lodgement and Subsequently obstruction may be at the "uretheral process".

5- If obstruction is total and U.B- not ruptured a "full bladder" may be detected by rectal exploration in cattle and by abdominal palpation in small ruminants.

b- U.B or urethary or rarely the kidney later ruptured, usually in 48 hours (it may take longer, particularly it the obstruction is not complete).

(B) Rupture of urethra:

l-Necrosis and Perforation of the urethral wall at the site of obstruction frequently occur.

2- Urine leak through the defect and accumulate in these s.c. tissue the Sheath and ventral abdominal wall 8 water belly phenomenomena.

N.B:

Water belly phenomenomena in feedlot ruminates is referred to the characteristic ventral swelling which is a specific manifestation of urethral rupture.

3- Aspiration from the swelling yields a clean fluid that may not smell of urine until heated.

4- examination reveals either collapsed or only partially distended bladder.

5-In advanced cases necrosis and gangrene with sloughing of ventral abdominal tissue may occurs.

(C) Rupture of the urinary bladder:

Rupture occurs most frequently on the dorsal side of U.B.

1- Rupture of U.B is the most serious sequel to urethral obstruction due to uremia.

2- Initial relief is the first signs of rupture of U.B and clear clinical signs for 1-2 days.

3- Uremia becomes severe next few days after U.B. rupture therefore the animal progressisses to severe depression, anorexia, and dehydration.

4- Ventral abdominal distention by urine may be noticed, and fluid may be detected by ballottement of the abdomen.

5- U.B not palpable via rectum, and the abdomen may feel empty.

Diagnosis:

I) History II) Clinical signs.

III) Lab. Diagnosis.

A- Blood analysis:

1- Bun, serum creatnine and plasma protein concentration raised. Serum potassium also elevated and hyperkalemic presents an anesthetic risk (due to cardiac abnormalities) in small ruminates if surgery is being considered.

2- Serum sodium and chloride values are depressed in ruminants with rupture of urethra and/or bladder.

3- Serum phosphate is the best prognostic indicator in cattle with ruptured bladder and that all steers with phosphate levels of greater than 9 mg/dl. Died.

B- Analysis of aspirated fluid:

1- Examination of fluid aspirated from the abdomen or subcutaneous tissues is difficult because of dilution with interstitial fluid, but heating may result in liberation of a urine odor.

2- Creatinine concentration of the aspirated fluid is usually more than double that of serum, so the ratio of peritoneal fluid creatinine to serum creatinine may be used disgnostically.

3- If the bladder has not ruptured but is severely distended, the creatinine and BUN concentration of the aspirated abdominal fluid may be only slightly higher than found in the serum.

4- Urea and creatinine diffuse from the affected areas into the circulation and sodium and chloride are drown into the area because of their low concentration in urine, causing a systemic hyponatremia and hypochloremia.

Treatment:

Aim of treatment is allowing animals to continue to gow enough to reach market weight:

1-A favorable response to treatment of urethral obstruction in feedlot cattle with aminopromazine has been reported, and the use of antispasmodics e.g: promazine or acepromazine in early cases allow sufficient relaxation of the urethra for the calculus to passed.

2- Surgical perineal urethrostomy proximal to site of obstruction is performed in cases in which the urethra has ruptured. The disadvantage of this surgery is the risk of stricture at the urethral incision.

3- Laportomy is performed and U.B repaired after cystotomy in cases in which the bladder has ruptured. A catheter (foley or mushroom type) can be placed in abdomen, and the urine drained slowly. Drainage must be with concurrent " fluid therapy" to normalize the animal metabolically. Fluids consisting of half-strength normal saline with 5% dextrose are effective in reducing the serum potassium concentration and hel, somewhat in restoring sodium and chloride concentration. The fluid from the abdominal activity, lowers the serum UN and creatinine.

Prognosis in steers is muss less favorable and only 50% can be expected to recover following surgery.

4- Surgical amputation of urethral process for treatment of obstructive urolithiasis is small ruminants to relive the obstruction can be reestablish the flow of urine.

In all cases the urethral process should be amputated to see if the obstruction can be relieved simply.

Essential medical therapy to normalize the metabolic status of the animal with obstructive urolithiasis.

Prevention:

1-Strutive urolithiasis in feedlot cattle controlled by ensuring that the phosphorus content of the cereal-based ration is portionately reduced by the inclusion of additional calcium.

- Alfa-alfa hay is an excellent source of calcium. Absorption of phosphorus from G.L.T is reduced by increasing calcium in the diet.

2- Gradual addition of salt to the diet of feedlot cattle up to level of 4% to 5% does not affect feed intake, but further reduces the incidence of urolithiasis.

- This is probably because of diuresis (due to increasing water intake) and dilution of urine, but the presence of additional chloride ions may lead to the formation of magnesium chloride in the urine, which is more soluble than magnesium phospnate.
- Chloride ions in urine are also capable of binding to mucorproteins, thereby decreasing silicalte and phosphate binding and diminishing calculus formation.

3- Availability of water at all times is essential.

4- Acidify the normally alkaline urine e.g: addition of ammonium chloride to the "diet", because struvite precipitates in alkaline solution.

- this reduces the incidence of urinary calculi in steers and in lambs when included at 0.5% of the ration.

EXAMINATION OF HERDS

(I) Exam. Steps

The necessary steps for exam. of a herd are include 4 steps:

Step 1: Defining the abnormality.

Step 2: Defining the pattern of occurrence.

Step 3: Defining the etiology group.

Step 4: Defining the specific etiology.

Step 1: Defining the abnormality

1- Defining the abnormality either clinical or subclinical.

2- In some cases the abnormalities will be obviously clinical e.g: clinical mastitis. or in other cases the abnormalities may be "complain" e.g lowered milk production.

Step 2: Defining the pattern of occurrence.

- Defining the pattern of the abnormality of the herd by examination of the available clinical, laboratory and production data for·

1- Time of occurrence e.g: season of year, age incidence of abnormality or

stage of pregnancy or lactation.

2- Genetic background of the abnormality e.g: animals belonging to which sive and dam groups .

3- Nutritional states of the farm e.g: existence of deficiency or excess nutrition

4- General management of the herd e.g: housing.

5- Vaccination history.

Step 3: Defining the etiology group.

Determine to which etiology category yhe abnormality most logically belongs e.g:

1- Infectious disease.
2- Medicinal disease.
3- Inherited disease.
4- Management disease.

N.B

In so many cases herd problems are not the result of a single error but are multifactorial, with several causes.

Step 4: Defining the specific etiology.

Make "herd diagnosis" by determination of the "specific cause" with the aid of:

1- Labortory diagnosis (Biochemical, microbiologyical, serological.....etc)
2- Diagnostic therapy (Response to treatment).
3- Response control measures.

(II) Techniques In Exam. Of the herd

Some techniques used in exam of group or herd of animals are:

A- Clinical exam.
B- Paraclinical exam. (Lab exam).
C- Numerical assessment of performance (statistical assessment of performance).
D- Response trial.
E- Complete exam of a herd.

I- Clinical exam.

1- Clinical exam is essential particularly if a clinical illness is a feature of the disease.
2- A representative sample of animals is examined and each submitted to a proper clinical exam.

3- Selection of the animals to be examined should include ten to twelve

animals as a minimum, and a large groups about 10% of the total number.

1- The groups should include eight sick animals, four advanced and four early cases and four normal animals as a control.

2- General clinical exam should carried out by exam of pulse, resp, temp, m.m, L.n mouth and skin plus intensive exam of the system suspected of involvement on the basis of the information provided by history e.g: A history of a herd problem of red urine in cattle would lead to a special clinical exam of the urinary system.

II- Paraclinical exam. (Lab exam).

1- The principle consideration in a herd exam is the need to collect samples at the same time that clinical exam done (paraclinical) then:

- Record the clinical observations.
- perform the clinicopthological lab exam.

2- The other consideration when planning a "sample system" is how the results will be interpreted in relation to normal standards.

3- It is usually safer to collect samples from:

- Normal animals.
- Animals not clinically abnormal but have exposed to the disease (animals in incubation period a subclinical stage).
- Disease animals.

N.B:

The number of animals to to be included usually six to ten in each group.

C- Numerical assessment of performance

*** Productivity index require to collect data over a period of time judge :**

1- The body weight in fatting farms.

2- Milk production in dairy farms.

3- Racing performance in horses and whether these parameter is going up or down.

* It is necessary to compare the results of productivity index with standards derived from comparable or peer farms.

* A computer program may be essential to evaluate the productivity index.

(D) Response trial

* In practical situation a diagnostic response trial can be performed by two treatments A and B or a combination AB and a control.

(E) Complete exam of the herd

* There are many circumstances in which some sort of herd surveillance is necessary .

* Some examples are:

1- When the diagnosis has been made and treatment or control measures stanted there is a need to monitor the results. This might entail measuring the percentage of animals that seroconvert after vaccination.

2- When a prevalence of a suspected disease is present e.g subclinical mastitis in diary:

- Herd entail periodic individual.
- Cow milk cell country.

3- Exam of herds not for the purpose of making diagnosis, but with the objective of prognostic indictor e.g; conducting Compton metabolic profiles in an attempt to predict of occur of a metabolic defining.

COMPTON METABOLIC PROFILE TEST

Definition:

It is a herd test based on the concept that laboratory measurement of certain components of blood will reflect the metabolic and nutritional status of the herd with or without the presence of clinical abnormalities.

Example:

Blood glucose mean lower than normal in a group of dairy cows indicates the presence of subclinicail or clinical Ketosis.

Aim of the test:

Metabolic profile test is a reliable test for:

1- Making a complete health service to a herd by early diagnosis of the metabolic and nutritional deficiency diseases.

2- Optimize livestock production by determination of the adequacy of the diet for cows to produce certain quantity of milk i.e obtain maximum yield at minimum costs.

3- Selection of superior individuals.

Procedure of the test:

(A) Blood samples are collected at least 3 times yearly:

1- Summer 2- Autumn 3- Winter

or when a metabolic or nutritional deficiency disease is suspected.

B) Blood samples are collected from 3 groups each of seven cows:

1- Dry cows

2-Medium-yield lactating cows

3- High yield lactating cows.

C) Blood sample must be collected at the same time of day at each collection.

D) Two blood samples are obtained from each animal:

1- 5ml in vials containing oxalate-fluoride for determination of glucose and inorganic phosphorus.

2- 20-30 ml in heparinied vials for determination of other components.

E) The following laboratory analysis have been done in Compton metabolic profile test:

1- Hemoglobin.

2- Packed cell volume (PCV)

3- Blood glucose.

4- Blood urea.

5- Total serum protein.

6- Serum albumin.

7- Serum globulins.

8- Serum calcium.

9- Serum inorganic phosphate.

10-Serum magnesium.

11-Serum sodium

12-Serum potassium. Occasionally

13- serum iron

14-Serum copper.

15- Plasma nonestrified fatty acids (NEFA).

Interpretation of results:

1-Low hemoglobin indicative of anaemia.

2- Low PCV indicative of anaemia.

3- High PCV indicative of haemoconcention and dehydration.

4- Low blood glucose (hypoglycaemia) indicative of subclinical and clinical Ketosis.

5- Low blood urea nitrogen (BUN) indicative of minimal protein intake.

6- High BUN (uremia) indicative of renal insufficiency.

7- Low serum total protein (hypoproteinaemia) indicative of law protein intake.

8- Low serum albumin (hypoalbumin) indicative of hepatic insufficiency.

9- Low serum globulins (hypoglobulin) indicative o: low immonlogical status of animals.

10- Low serum calcium in late pregnant or early lactation indicative of subclinical or clinical milk fever.

11- Low serum inorganic phosphorous (hypophosph.) indicate subclinical post-parturient haemoglobinurea or indicate milk lamenness (osteomalacia).

12-Low serum magnesium during lactation indicates subclinical or clinical hypamagnesium.

13-Low level of serum sodium (hyponatremia) in early lactation in cows particularly in summer may be associated with depraved appetite.

14-Low level of serum - K (hypokalemia) indicative of creeper downer cows (recumbency).

15-Low level of iron indication of iron deficiency anaemia.

16- Low level of copper indication of falling disease in cattle, enzootic ataxia and sway back disease in sheep. -

17-Free fatty acids more sensitive than glucose as an indicator of energy status of lactating cows except during early lactation (due to excess variability of this relationship during this stage).

بسم الله الرحمن الرحيم

METABOLIC DISEASES

(Metabolic disorders, Metabolic pathology, production diseases, food animal production diseases)

Introduction:

1- Metabolic diseases usually associated with pregnancy, parturition and lactation.

2- Metabolic diseases usually associated with high productivity and reproductivity of the livestock animals.

3- High productivity e.g.: More than 20 Kg./day in Holstein-Friesian cows (total cost of H.F. cow=14 kg milk/day.

-High reproductivity e.g.: Animals carry more than one fetus (give twins, triples...etc. e.g.: ewes and goats).

4- Metabolic diseases of our native breeds is mainly attributed to "nutritional faults", more than associated with high productivity and reproductivity.

e.g:- Egyptian native breed balady cow give 700-900 kg/year.

- Egyptian buffaloe give up 1200 kg milk per year.
- Domiaty cows give up 1200 kg milk per year.
- Fresian cows give 4000-5000 kg milk/year.
- American Holestien cows give 6000-7000 kg/Year.

5- The economic definition of the cow is udder plus uterus and 60% of milk production from posterior 2 quarters of udder.

6-Metabolic disorders countered in food animals are, milk fever, Hypophosphataemia, Hypomognesaemic tetany, Ketosis, and fat Downer cow syndromes.

(1) MILK FEVER

(Parturient paresis, parturient hypocalcaemia)

Milk fever is a misnomer because it is not a febrile disease.

Definition:

Milk fever is a metabolic neuretic disease occurring most commonly about the time of parturition and characterized clinically by generalized paresis, circulatory collapse and depression of consciousness and biochemically by hypocalcaemia.

Incidence, occurrence and predisposing factors:

1- Cattle most susceptible animals (particularly Jersy breed 30%}.

2- The disease may occur sporadically in buffaloes, sheep;and goats (no records about the disease in camels).

3- Aged or senile cattle mostly susceptible and age incidence 5-10 years (3-8th calving).

4- Heavy milkers cows (20 kg/day and more milk production).

5- Usually 24 hours before or mostly 72 hr. post. partum due to sudden evacuation of colostrum. (125 mg% in cow).

6- Incidence in winter more than other seasons.

7- High protein diet before calving predispose to the disease.

8- Undue fatigue and excitment predispose to the

Etiology:

1- The exact cause of milk fever is unknown.

2- The known is that the basic biochemical finding in the disease is hypocalcaemia.

3- How the hypocalcaemia developed?

a) At calving, there is a normal physiological fall in blood calcium. But a more significant fall occurs in cows, which develop the disease.

b) There are 2 factors control-beside the intrinsic endocrine factor calcium haemostasis (blood calcium pool) in the body

1- calcium inflow

2-calcium outflow

Decreased calcium inflow: (due to)

1- Impairment of calcium absorption from gut due to bowel stasis during partition.

2- Insufficient mobilization of calcium from bone due to aging or senility.

Increased calcium outflow: (due to)

1- Excessive loss of calcium in colostrum.

2- Excessive calcium excretion in faeces via gut due to for example profuse diarrhea.

3- Increased calcium bone mineralization.

4- increased calcium excretion in urine due to diuresis.

Other theories suggested in the pathogenesis of the disease:

1- Parathyroid dysfunction which lead to guanidine amino acid intoxication which inturn result in hypocalcaemia.

2- Thyroid dysfunction (which secrete calcitonin hormone from C-cells) as histological section of C-cells of the gland found to be depleted

3- sex harmons, as oestrogen antagonist to parathyroid.

Other biochemical changes recorded with the constant hypacalcaemia of paretic cows:

1- Hypophosphataemia -- due to parathyroid dysfunction.

2- Hypo-or hypermagnesaemia.

3- Hyperglycaemia:

Hyperglycaemia (80-90 mg%) usually accompany milk fever and severe hyperglycaemia (up to about 160 mg%) occur with severe paresis arid the cow become diabetic-like, due to inhibition of insulin release from B-cells of pancreas under the influence of hypocalcaemia.

Clinical signs:

3 stages of the disease are known including:

A) prodromal stage (excitative short phase)

B) Stage of sternal recumbency (semicomatosed stage).

C) Stage of lateral recumbency (comatosed stage).

(A) Prodromal stage: (excitative short phase)

Characterized by:

1- Excitement and restlessness (vigrous-licking of skin).

2- Hypersensitivity and fine muscle tremors.

3- Disinclination to eat or move.

(B) Stage of sternal recumbency (Semicomatused stage):

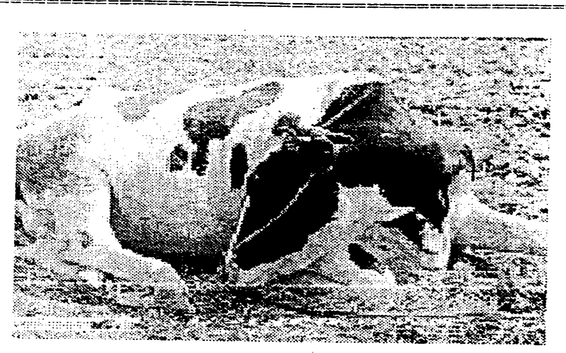
Characterized by:

1-Depression of consciousness.

2- Animal in sternal recumbency and the head turned laterally toward flank region resting on the shoulder region.

3- Dry muzzle dilated pupile cold extremities and loss of anal reflex, as well as, atonic digestive tract manifested by constipation.

4- Temp. normal or even subnormal.



Characteristic position in milk fever

(C) Stage of lateral recumbency (comatosed stage):

Characterized by:

1- Animal almost comato2ed.

2- Animal in lateral recumbency,

3- Complete flaccidity of limbs and loss of nervous reflexes.

Diagnosis:

I) History.

II) Clinical signs.

III) Diagnostic therapy: Estimation of serum calcium level usually below 6 mg %, sometimes, as low as, 3 mg % (normal around 10 mg %).

IV) Diagnostic therapy: (therapeutic diagnosis)

Milk fever is a dramatic disease, due to treatment with calcium borogluconate usually followed by a dramatic recovery within few minutes.

Treatment:

Most untreated cases die within 1-2 days.

(A) Nursing care:

1- Stomach tube feeding to avoid aspiration pneumonia.
2- Turning of animal from side to side to avoid tympany.
3- Massage of limbs and bony prominants to avoid limb dysfunction.

(B) Chemotherapy:

Calcium borogluconate is the drug of choice or the classical **treatment.**

400-800 ml C-B-.G 25% slow I/V or S/C repeated after 12 hrs.if no r response, then repeated after 24 hrs. And if no further response use alternative therapy containing phosphorus and magnesium e.g calphomag (Virbac).

(C) Udder inflation:

Old method (1897) and not usually recommended due to inducing injury and mastitis.

Prevention:

1- Feeding high phosphorus low calcium ration in two last months of pregnancy (dry period).
2- Single I/V or S/C-10 million I.U crystalline vitamin-D 8-10 digs before calvinng repeated if the cow not calving.
3- Diet rich calcium after parturition only.

****

(II) METABOLIC HYPOPHOSPHATAEMIA

(Post-parturient hemoglobinuria in cows)

(Hemoglobinuria of pregnant buffaloes)

Definition:

It is a metabolic haemolytic disease of lactating cattle, and pregnant buffaloes, characterized clinically by hypophosphataemia and intravascular haemolysis of erythrocytes.

Incidence, occurrence and predisposing factors :

1- The disease affect mostly cattle and buffaloes.

2- The disease occur 3-4 weeks after parturition in cows. While occur in "mid-ten." gestation period in buffaloe.

3-The disease mostly affect aged and senile cattle and buffaloes and age incidence usually between 5-10 years old.

5- Heavy milk production predispose to the-disease.

6- Feeding plants or rations which are normally low in phosphorous and high in calcium content, like feeding Green Barseem (Alfa alfa or clover) exclusively for long period may extend up to 4-6 months predispose to the occurrence of the disease particularly in Egypt (Beest, Turnips, Kale in Europe).

7- The disease have a seasonal occurrence in Egypt usually associated with late spring (April-May).

Etiology:

1- The exact cause of the disease is "unknown"

2- The basic biochemical findings in the disease is low inorganic phosphorous level in blood of affected animals (hypophosphataemia).

3- Hypophosphataemia may develop due to "inadequate" intake of phosphorous in ration which may be exacerbated by ingestion of high calcium in ration.

N.B

The cause of intravascular haemolysis of erythrocytes is "unexplained", but may be attributed to:

a) Increased fragility of red cells due to inadequacy of phosphorous for formation of phospholipids member of erythrocytes.

or

b) Haemolytic factors of "saponins"" present in Alfa Alfa leaves (e.g of haemolytic factors are thiocynate, nitrate and sulfoxides toxins).

These toxins causes irreversible oxidative changes in haemoglobin, leading to formation of what is called Heinz-bodies.

Red cells which containing these H.R. are removed by spleen for haemolysis.

Clinical signs:

1- The animal voiding "dark red-brown" to almost "black" urine.

2-The animal eat and milk normally for 24 hours after the appearance of hemoglobinuria.

3- Pale m.m., or even ecteric m.m. in severe cases (due to haemolytic jaundice).

4- Tachycardia and increased pulse, and respiration rates above normal ranges.

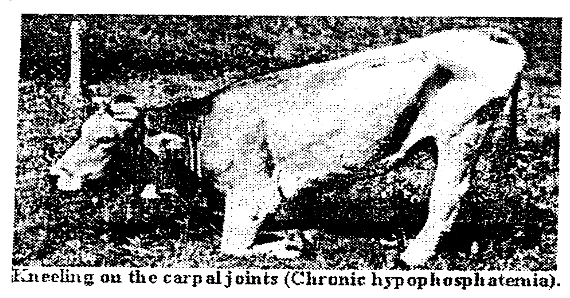
5- Normal rectal temperature.

6- Recumbency after an acute course of 3-5 days.

7- The disease slowly recovered as convalescence is prolonged for up 3-4 weeks (chronic cases) and pica is often observed during this stage.




Yellow staining of the conjunctiva and valva MM in severe cases of hypophosphatemia.



Kneeling on the carpal joints (Chronic hypophosphatemia).

Diagnosis:

(I) History

(II) Clinical signs

(III) Laboratory diagnosis:

(A) Urinanalysis:

1- Benzidine test for detection of blood in urine (+ve)blue.

2- Centrifugation of urine sample --- > persistant red column

Haememoglobinurea

(B) Blood analysis:

1- Estimation of blood haemoglobin ----> drop to mg% (N=4-7 mg%)

2-Estimation of blocd haemoglobin --->drop to 5 gm% (Normal 10-12 gmo)

3- Determination of total erythrocyte count --->drop to 2 millions (Normal 5-8 millions)

4- Determination of PCV (Haematocrit value ------ > drop to 2 5 volume %(N= about 35)

N B

Estimation of serm bilirubin and blood urea......> raised.

- The disease must be differentiated from other causes of haemoglobinurea e.g Bebesiosis leptospirosis, clocstridium haemolyticm , copper poisoning, creosol poisoning, ,onion poisoning, onion poisoning and water intoxication.

Treatment:

1- Change the diet which is rich in calcium by another diet rich in phosphorous e.g bran instead of barseem.

2- Elevation of serurm inorganic phosphorous level by:

a) I/V injection of 60 gram Na acid phosphate in 300 dist. water. Followed by:

b) S/C of similar dose ; times with 12 hours intervals. Followed by;

c) Oral similar dose till disappearance of haemoglobinurea.

3- Bone meal 120 gram twice daily till disappearance of haemoglobinurea (but expensive treatment)

4- I/V calcium hypophosphate in glucose solution (prepared by dissolving 30 gram in 10% glucose).

5- The use of commercial useful preparation e.g: Tonophosphan (Hochest) 20 ml I/V or I/M till disappearance of haemoglobinurea.

- Phospho-20 (Verbac) 15 ml of haemoglobinurea

*** Prevention of the disease:**

Aged or senile lactating cows, or pregnant buffaloes must given -as a prephylaxis half the therapeutic dose of sodium acid phosphate (30 pm) or bone meal (60 gm).

****

LACTATION TETANY

(Hypomagnesaemic tetany , grass tetany, grass stuggers)

Definition:

Highly fatal metabolic diseases of lactating ruminants, characterized clinically by hyperaesthesia, tetany and convulsions and biochemically by hypomagnesaemia.

Incidence, occurrence and predisposing factors:

(A) Incidence:

(a) Species incidence : Cattle (sometimes sheep).

(b) Age incidence : 7-10 years old (5-8 Lactation).

(c) Time incidence : 2-4 months after parturition.

(B) Occurrence:

(a) Lactating cattle grassing on high potassium spring lush pastures and green cereal crops (High potassium levels decreases Mg. Absorption).

(b) Lactating cattle fed diet intoxicated with potassium fertilizers or urea (reduces availability of soil magnesium).

(C)predisposing factors:

(a) Starvation : 24-48 hours depress serum magnesiums significantly.

(b) Diarrhea : Reduce magnesium absorption from intestines

(c) Cold weather stress : Increase urinary excretion of magnesiums.

(d) Long transport: Depress serum magnesium and calcium.

Etiology:

- Basic biochemical finding in lacation tetany is hypomagnesaemia 1-2 mg% (N 2.5 – 3).

N.B:

Why tetany occurs?

- Calcium ions activator for acetylcholine responsible for muscle contraction.
- Magnesium ions activator for cholinesterase responsible for Muscle. relaxation.

Clinical signs:

(A) Acute form:

1- Sudden onset of hyperaesthesia and muscle twitching .

2- staggering in gait followed by falls down with tetany and convulsions.

3- During episodes (attack) there are:

a) Opithotonus (back-head).

b) Pricking of ears.

c) Nystagmus (Rotation of eye ball).

d) Retraction of eye lids.

e) Champing of jaw.

f) Frothing at from mouth.

4- Between episodes animal lie quiet but any noise or touch starting other attack .

5- Pulse and respiration accelerated.

6- Temperature moderatly elevated (due to muscular spasm).

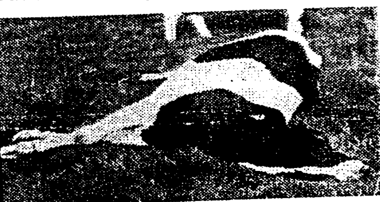
7- Death from respiratory failure.

(B) Subacute form :

Same signs of acute but onset gradual and course longer.

(C) Chronic form :

Animal have low serum magnesium but shows no symptoms.

Opithotonus (hypomagnesemia)

Diagnosis:

(A) **History:**

(B) **Clinical signs:**

(C) **Laboratory diagnosis:**

(a) Serum magnesium level commonly between 1-2mg % (normal 2.5-3 mg%).

(b) Low urine magnesium level.

(c) Low CSF magnesium level.

(d) Bone biopsy from ribs commonly revealed disturbed Ca: Mag. ratio.

(D) Diagnostic therapy (Therapeutic diagnosis):

Diseased animals respond well to calcium-magnesium therapy.

Treatment:

- contraindicated to use Mg compounds alone (cause cardiac arrest).
- Safe therapy to use combined calcium-magnesium preparations as follow:

1- Calcium borogluconate 15% (I/V 500 ml).

2- Followed by: Magnesium lactate 15% (S C 250 ml).

3- Followed by: Magnesium sulphate (oral 125 gram).

- Preparations: Cal-D-Mg (Pfizar). Calphomag (Vibrac).

  N.B: Tranquilizer e.g: Chloropromazine Hcl (Neurazine) used and useful before calcium-magnesium therapy in severe cases.

Prevention:

1- Magnesium supplementation of diet with crude magnesium (calcined-magnesite)60 gm/head which can be mixed with molasses:

2- Magnesium Bullets placed in reticulum for slow liberation of constant traces of magnesium daily for long period as long as several months or even years.

* * *



(IV) KETOSIS

Bovine ketosis. ----------> Acetonaemia

Post-parturient dyspepsia

Definition:

Ketosis is a metabolic disease of ruminants characterized clinically by nervous and or digestive disturbances and biochemically by low hepatic glycogen, hypoglycaemia, ketonemia and ketonuria.

Incidence, occurrence and predisposing factors:

1- The disease occur in:

a) Lactating cattle and buffaloes.

b) Pregnant sheep and goats.

2- Most incidence occur in:

a) 1st month of lactation in cattle and buffaloes.

b) Last month of pregnancy in sleep and goats.

3- Incidence usually in :

a) Heavy milkers cattle and buffaloes

b) pregnant ewes carry more the one lamp (ewes pregnant in twins, triplets .. etc. due to restriction of rumen volume the developing foeti, or due to inadequate et intake).

4- Aged senile cattle, buffaloes, sheep and goats are mostly susceptible and age incidence 5-10 years.

Etiology and pathogenesis:

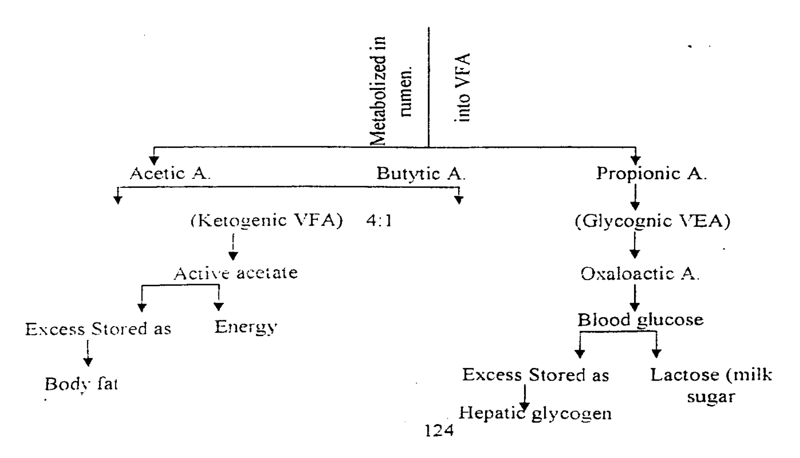
1- The basic biochemical findings in ketosis is hypoglycemia.

2- Hypoglycaemia may occur due to feeding of lactating cows and pregnant ewes diets of low caloric content.

3- Feeding diets of low caloric content, lead to impairment of normal carbohydrate metabolism follow:

a) Feeding diets "sufficient is carbohydrate content requirement for ruminants lead to the following biochemical pathway:

Ingestion sufficient carbohydrate



b) Feeding diets insufficient in carbohydrate content requirement for ruminants lead to the impairment in this normal for carbohydrate metabolism resulting in hypoglycemia and ketonaemia as following biochemical abnormal pathway.

*** How hypoglycemia developed?**

- Insufficient carbohydrate ingested ⟶ insufficient propionic VFA in rumen ⟶ insufficient oxaloacetic F.A ⟶ insufficient blood glucose ⟶ hypoglycemia.

*** How Ketonemia and Ketonuria developed?**

- Insufficient carbohydrate ingested--> Active acetate converted into:

Ketone bcdies = (1) + (2) + (3)

1-Acetoacetic acid

2- Acetone

3- Beta-hydroxybutyric acid.

Endocrine and metabolic mechanisms suggested in the pathogenesis of Ketosis: (Bovine and Ovine)

(A) Thyroid dysfunction: This proved by:

1- Low level of protein bound iodine (PBI) in serom of affected lactating cows.

2-Iodine and thyroprotein administration have a preventive and curative effect in ketotic cows.

(B) Adrenal dysfunction: This proved by:

Elevation of plasma cortisol level in blood of pregnancy toxaemic ewes.

N.B.

Recently, plasma cortisol level raised when animals putted under any stress (nutrional, environmental.. etc.) then cortisol destructed by the detoxification mechanism of the liver if healthy: Cortisol still raised in

ewes suffering from pregnancy toxaemia due to ill-health liver (fatty infiltration of liver).

(C) Feeding ketogenic diets:

Feeding Ketogenic diets (ration rich in protein) leads to excessive production of ketogenic VFA (acetic A., and Butyric A.), which are the precursors of ketone bodies.

(D) Deficiency of certain trace element:

Cobalt deficiency in the diet may predisposing for ketosis due to its role in "metabolization of propronic acid to oxaloacetic acids.

Clinical signs:

1- Two forms of the disease known in bovine ketosis (digestive form and nervous form).

N.B: Sometimes there is mixed form (nervous & digestive signs in the same animal)

2- In both forms, there is sweet smell in breath and during milking in ketotic cows.

(A) Digestive form: (Woody cow)

a- Gradual loss of appetite and rapid loss of body weight.

b- Decrease or absc2nce of ruminal movement.

c- Rumino intestinal stasis, manifested clinically by constipation.

(B) Nervous form:

Nervous form may he attributed to "Isopropyle alcohol" which results from further destruction of acetoacetic acid.

a-The cow staggering in gait, walking in circle with crossing of legs and pushing head into corners as be blind.

b- Hyperesthesia, tremors, tetany and convulsions and terminal paresis and coma (recumbency).



Paresis of the hind quarter in a cow with nervous ketosis

Diagnosis

I) History of the disease.

II) Clinical signs.

III) Lab. Diagnosis: Urine analysis (urine analysis) Milk analysis

Blood analysis

1- Detection of acetone in urine and milk by modified rother's test or Strip test.

2- Determination of blood glucose level Below 40 mg% (Normal 40 – 60 mg%)

3- Determination of total ketine bodies levels up 1000 mg% (normal up10 mg)

4- Blood acidosis (metabolic toxaemia), and raised NPN may present.

* What's physiological acetonaemia?

* What's secondarily ketosis?

Treatment:

The arm of the treatment is elevation of blood glucose level by :

1-I/V adm. Of 500 ml 40-50% Dextrose for cows N.B:

Recent theory this method five, a transient relief, some recommended oral glucose after closure of esophageal groove by vasopressin in hormone.

2-oral glucose forming substances e,g:

a- Sodium propionate or lactate 120 – 240 gm daily.

b- Starch 30 gm + chloral hydrate for digestion of starch + molasses.

3- Synthesis is glucose from non-carbohydrate Origine by physiological process of gluconegensis using some hormones e.g : ACTH, corticos teroids and glucocorticordes (Dexamethazone 5-20 mg/ head or 10-30 mg Betamethazone/head for ketotic cows)

4- Cobalt or B_{12} may be useful.

5- Propylene glycol 125-250 gm orally given twice daily for ketotic cows with salt mixture containing cobalt.

Prevention or control:

Lactating cows and buffaloes and pregnant ewes and goats must take ration sufficient in carbohydrate requirement for asufficient caloric intake and subsequently avoidance of disturbances in carbohydrate metabolism and VFA which leads to hypoglycaemia and ketonaemia .

****

FAT COW SYNDROME

Synonyms

- Fat cow Ketosis
- Pregnancy toxemia in cattle
- Fatty infiltration of liver.

Definition :-

Fat cow syndrome is a sporadic metabolic neuretic disease of pregnant beef cattle characterized by low morbidity (1-3%) and high motality (100% fatality).

Incidence, occurrence and predisposing fators:

1- the disease affect fat or obese cows.
2- Mostly pregnant beef cattle .
3- Commonly in late pregnancy period (last 2 months of pregnancy).
4- Mostly pregnant with twins.

5- Commonly in the first-calf heifers than older cows.

6- In cows that have become fat or obese because of heavy feeding in early pregnancy, then subjected to severe "nutritional stress" during the last 2 months before parturition.

Etiology :

1- Mobilization of excessive quantities of fat from body depots (particularly S/C fat) to the liver due to deprivation of feed i.e the disease occur due to state of severe nutrional energy deficiency .

2- The disease is considered as an exaggerated from of ketosis in pregnant beef obese cows.

Clinical Findings :

Affected cows are invariably fat and in the late stages of pregnancy .

1- Affected cows are depressed for 10-14 days.

2- Suddenly become completely anorexic.

3- Transitory period of restlessness, excitement ,incoordination, stumbling gait and aggressive-ness.

4- Sternal recumbency.

5- Skin on muzzle is dry, cracked and may peel off.

6- Feces are scant and firm, terminally become soft diarrohic and yellow orange, but still small in volume.

7- Tachycardia and increased pulse rate, and respiration rapid and accompanied by respiratory grunt and clear nasal discharge .

8- The cow become comatose and disquietly after a course of the disease about 10-14 days.

Diagnosis:

I) History II) Clinical findings

III) Laboratory diagnsis :

1- Hypoglycaemia (but terminally blood glucose level are often raised to high level).
2- Marked ketonemia and ketonuria.
3- Elevated liver function tests (bilirubin and hepatic enzymes) .
4- Proteinuria (due to fatty infiltration of kid.).

IV) Liver biopsy :

To determine the severity of the fatty liver by 2 methods :

1- Biochemically by estimation of liver triglycerides.
2- Histopathologically by estimation of lipid content of liver.

Treatment :

1- In general, cows which are totally anorexic for 3 days or more or recumbent treatment is ineffective and will die.
2- There is a slight transitory response to pare-nteral treatment with glucose, glucocorticoids, cobalt and B_{12}
3- Intensive therapy must directed to correct the pathophysiological effects of the disease and fatty liver by :
    a- Continuous I/V infusion of glucose electro-lyte soln.
    b- Intraruminal adm. Of rumen juice (5-10 Litres) from normal cows to stimulate the appetite of the affected cows.
    c- Oral adm. Of propylene glycol to promote glucose metabolism.
    d- S/C injection of insulin (zinc protamine) 200-300 I.U twice daily to promote peripheral glucose utilization.
    e- S/C injection of choline chloride 25 gm every 4 hours (in severe cases).
    f- The use of anabolic steriods e.g. Vebenol 300 mg or trenbolone acetate.

g- Water and balanced electroiytes (10-30 liters) can adm. Intraruminally.

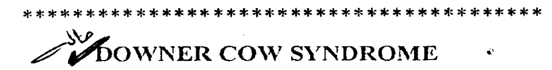
Control :

Because of the large economic losses associated with pregnancy toxemia in cattle :

1- Prevent pregnant beef cattle from becoming fat during the last trimester of pregnancy .

2- Apply metabolic profile test as mean of assessing energy status by estimation of both glucose and B-hydroxy butyric acid.

****



DOWNER COW SYNDROME

Definition:

One of the major problems dairy cattle practice is the so-called Downer cow syndrome in which the aged animals goes down most commonly during pregnancy and in periparturient period.

Etiology:

A- The cause is largely unexplained and the syndrome rarely in animals other than ruminants.

B- A number of metabolic disterbances, serios infections diseases and injuies to the muscle-skeletal system occur at the be a factors, but for alack of abetter classification, these cases are grouped and described under the heading Downer cow syndrome.

C- Generally the term Downer usually applied to animals that are down and can not get back on their feets due to four major causes of several possibilities as follow:

I- Metabolic disturbances and nutrional deficiency diseases:

(A) Metabolic Disorders

1- Calcium in partureint hypocalcaemia or partureint paresis (milk fever)

2- Phosphorous in post-partureint haemoglobinuria or hypophosphataemia of buffaloes.

3- Magnnesium in Hypomagnesaemic tetany or lactation tetany .

4- Sugar in Ketosis or acetonaemia of cattle

5- Fat in fat cow syndrome (pregnancy toxaemia of cattle).

(B) Nutrional deficiency diseases:

1- calcium , phosphorous and vitamin – D deficency in osteomacia (milk lameness).

2- Traces elements defeciencies and disturbances minor electrolytes :

a) Copper in falling disease in cattle

b) Cobalt in bovine ketosis.

c) Potassium in Hypocalaemia-particularlly in the so-called Creeper Downer cows which are alert and crawl but unable to rises .

d) Protein deficiency in Hypoproteinaemia particularlly if accompanied by unspecific digestive disturbances.

II- Non infectious medicinal diseases:

1- Sever cases of rumen acidosis (due to rument histminosis).

2- Liver cirhsis (due to imairment of liver function especially the detoxification and inactivation of steroid hormones particularly oestrogen).

3- Liver absesses (due hyper or hypoglycaemia).

4- Last stages of pneumonia (due to hypoxia).

5- Last stages of pyelonepheritis (due to hypoxia).

6- Acute circulatory crisis (due to acute heart failure or acute circulatroy failure which both lead to brain ischaemia).

7- Traumatic pericarditis (due to generalized toxaemia).

8- Encephalopathy (encephalitis, meningitis, meningoencephalitis or encephalomalacia).

9- Sever thirst (due to haemoconcentration and hypoglycaemia).

10- Sever thirst (due to haemocncentration and dehydration .

11- Leukaemia.

12- sun stroke (over heating).

III- Infectious diseases:

Generally the last stages of any acute febrile infectious disease can cause recumbency, for instance:

A)Anthrax B) Black leg c) Malignant oedema

D) Mucosal disease E) Acute mastitis (due to toxaemia).

F) Generalized T.B. (due to tuberculous meningitis).

G) Last stage os tetanus (due to generalized tonic spasm).

IV- Surgical and obstitrical causes for recumbency:

Generally injuies to the locomotor system- musclo-skeletal-system (nerves, muscles, joints and bones) can causes in some cases recumbency:

1- Bones-- Broken bones (especially vertebral column and pelvis).

2- Joints -- Arthritis (especially knee or hock joints).

3- Muscles -- Ischamic muscle necrosis or muscular rheumatism.

4- Nerves: pressure on nerves (especially obturator nerve and this usually causing recumbancy in the last third of pregnancy if too large foetus is intrauterine in present).

Also nerves injuries (due to paresis or paralysis).

* Acute endometritis (due to generalized toxaemia).
* Uterine torsion (due to rupture of uterine artery).
* Uterine rupture (due to diffuse peritonitis).

Clinical findings:

(A) Typical cases :

1- Affected animals usually show no sings of illness until go down >

2- Bright and alert and make frequent attempts to rise, but unable to completely extending their legs (sternal recumbency).

3- Eat and drink moderately well and defecation and urination normal.

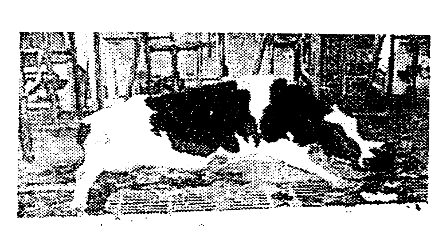
4- Temperature usually normal, somtimes elevated respiration unaffected and pulse usually weak .

(B) A typical cases:

1- The signs may be more marked and include a tendency to lie in lateral recumbancy with the head drawn back.

2- Still more severe cases, affected animals may show hyperaesthesia and sometimes tetany, os limbs.

3- These more severe cases do not usually eat or drink.





Downer cow syndrome

Diagnosis:

1-History. 2- Clinical signs.

3- Laboratory diagnosis.

Depends mainly on the suspected cause of recumb-ency.

- CPK and SGOT are usually elevated.

- Proteinuria or even myoglobinuria may be observed due to muscle damage from prolonged recumbency and muscle damage attributed to ischemia of muscles.

How to approach such cases:

(Nursing and chemotherapy)

A- Nursing care :

Recumbent animals especially bovine must be :

1- Frequent turned from side to side (to avoid tympany, M necrosis and para-analgesia which result from prolonged recumbency).

2- Provided with ample (large) bedding, green food and free water, as well as, not left, the cow on slippery ground surface (to make effort to rise).

3- Massaging of limbs may be successful especially for muscles of thigh, as a type of physiotherapy.

4- Slinging, but usually unsuccessful unless the cow is partially able to get her own.

B- Chemotherapy:

Depends mainly upon the clincal signs, biochemical finding and the most suspected or confirmed cause or causes. But the most satisfactory treatment program include the parentral injection of :

a- Electrolytes solutions containing calcium, phosphorous, magnesium, copper, selenjum, cobalt, and potassium.

b- Nutrients preparations containing dextrose and aminoacides.

c- Ployvitamins preparations containing vitamins A,E,D,B, (Thiamin) and B_{12} (cynocobalamine).

d- Anabolic hormones may be useful such as steroid gonadal hormones or other anti-inflammatory hormones like

corticosteroids or cortison derivatives particularly if toxaemia suspected (cortigen B_6).

e- Fluid therapy by oral or parenteral route is indicated for cows which not be drinking anormal amount of water.

Prevention or control :

Insufficient is known of the etiology for suitable control measures to be recommended.

N.B:

Some textbooks, mentioned that, the "downer cow syndrome" is condition occurs in cattle usually following milk fever when the cow still in prolonged recumbency even after two successive treatment with calcium borogluconate (C.B.G.). At necropsy there is traumatic injury to limb muscles and nerves, ischemic necrosis of limb muscles, myocarditis (may due to repeated injection of C.B.G.) and fatty infiltration and degeneration of the liver .

Etiology not clear, but the disease suggested to be a complication of milk fever.

Traumatic injuries to nerves of limbs are present in 25% or more of downer cows. Sciatic and obturator nerves are vulnerable to injury by pressure from oversized calf before or during parturition leading to obturator paralysis.

Overlong delay in treatment of milk fever and prolonged recumbency (more than 4-6 hours) is considered to be an important cause of downer cow syndrome due to ischemic necrosis of limb muscles and nerves.

Serum electrolyte imbalance or deficits have been suggested as a cause of prolonged recumbency (downer syndrome) following milk fever e.g, hypokalemil with hypophophatemia in " Creeper downer cows".

NUTRITIONAL DEFICIENCY DISEASES

1- Vitamin A deficiency.
2- Vitamin E + selenium.
3- Vitamin D + calcium + phosphorous :
   a) Rickets.
   b) Osteomalacia.
4- Hypomagnesaemic tetany of calves.
5- Trace elements deficiency :

a) Copper deficiency.
b) Selenium deficiency.
c) Zinc deficiency
d) Iodine deficiency

Diseases Caused By Nutritional Deficiencies include:

Diseases Caused by:

I) Deficiency of fat-soluble vitamins.
II) Deficiency of minerals nutrients.

Diseases caused by deficiency of fat soluble vit.

VITAMIN A DEFICIENCY
(HYPOVITAMIOSIS-A)

Etiology:

Vitamin-A deficiency occurs either:

1- As a primary disease due to absolute deficiency of vit. Aor its precursor carotene in the diet.
2- Or as a secondary disease in which the dietary supply of the vitamin or its precursor is adequate, but their digestion or metabolization is interfered.

Epidemiology:

1- Primary vit. A deficiency occurs most commonly because of lack of green or failure to add vit. A supplements to deficient diets.

2- Young calves and lambs depend mainly on their dam's ,ilk (colostrum) for their early requirements of the vitamin which is in higher conc. in colostrum.

3- Carotene or alcoholic form of vitamin A in green feed, does not pass placental barrier, therefore hepatic stores of vit. A in newborn calves and lambs nearly absent if carotene or alcoholic form of vit, A only Fed during pregnancy to their dams.

However, vit A in ester form in fish oils (injectable preparation) can pass and cause an increase in vit. A store in fetal livers.

4- Carotene and vit. A are readily oxidized in the diet in the presence of unsatuated fatty acids.

5- Heat. Light and mineral mixes increase rate of destruction of vit. A in commercial preparation.

6- Vit. E and C prevent loss of vit. A in feed stuffs and during digestion.

7-Continued ingestion of mineral oil (paraffin oil) as a preventive measure against bloat, causes severe depression of plasma carotene and vit. A.

8- Secondary vit. A deficiency may occur in case of chronic diseases of intestines and liver (site of storage of vit, a).

Pathogenesis:

(A) Night vision:

Vit. A is essential for regeneration of the visual purple necessary for vision in dim-light.

(B) CSF pressure :

1-Vit. A deficiency causes an increase in CSF pressure due impaired absorption of arachnoid villi and thickening of C.T. matrix of dura matter.

2- Increases (SF pressure causes convulsions and syncope.

(C) Bone growth:

Vit. A is essential for normal bone growth as its responsible for maintain normal position and activity of osteoblasts.

Therefore, hypovitamions is A causes incoordination of bone growth e.g.: facial paralysis and blindness due to construction of facial and optic cranial nerves are typical examples of this phenomenon.

(D) Epith. Tissue:

Vit. A is essential for normal epith. Tissue and its deficiency leads to atrophy of all epith. Cells particularly epith. Secretary cells, and these secretary cells are gradually replaced by stratified, keratinizing non secretary cells.

Examples of organs and systems be affected are, eyes, salivary gland, thyroid gland, urogential trace particularly placenta.

(E)Embryological development:

Vit. A essential for organ formation during growth of foetus in pigs and rats only.

Clinical findings:

(A)Night blindness:

Inability to see in dim- light.

(B) Xerophthalmia:

Thickening and clouding of cornea.

(C)Changes in skin:

1- Heavy deposits of bran-like scales on skin of cattle.

2- Dry, scally hooves with multiple, vertical cracks in horses.

(D) Body weight:

Emaciation and stunted growth occur only under experimental condition of severe vit. A deficiency but not occur under natural condition.

(E) Reproductive efficiency:

1- In male " retained libido" and degeneration of germinative epith. Of sonmiferous tubules causing reduction in number of motile, normal spermatozoa.

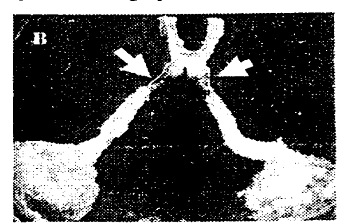
2- In female abortion due to placental degeneration and birth of dead or weak young plus retention of placenta.

(F) Nervous sings:

3-Total blindness of both eyes due to construction of the optic nerve canal (manifested by absence of menace reflex).

4- Encephalopathy, manifested by convulsive seizures due to increased C.S.F. pressure in beef calves at: 6-8 months of age. Affected calves may collapse (syncope) and may die during episode.





Vitamin A deficient cow with newborn calf (figure A). The calf was born blind and weak. The optic nerves removed from the calf are pinched at the point where they pass through the optic chiasma due to improper osteal growth (arrows).

(G) Congenital defects:

5- Congenital blindness of calves due to optic nerve construction.

(H) Other diseases:

6- Increased susceptibility to infection.

7- Otitis media and enteritis.

Diagnosis:

(A) History

(B) Clinical signs.

(C) Lab. Diagnosis:-

A-Estimation of plasma and liver vit. A and carotene:

1- Plasma carotene vary largely with diet. In cattle level of about 150 µg/dl is optimum.

2- Plasma vit. A conc. Ranged from 25-60 µg/dl in cattle.

3- Hepatic level of carotene ½ -4 µg/gm liver.

4- Hepatic level of vit. A 2-60 µg/gm liver.

B-Estimation of C.S.F. pressure :

- C.S.F is a sensitive early indicator of vit. A status.
- In calves, normal C.S.F pressure of less than 100mm of water but abnormal more than 200mm.

C- Conjunctival smears:

- Increased number of cornified epith. Cells.

Treatment:

Generally in hypovitaminosis – A, animals should be treated with vit. A at dose rate equivalent to 10-20 times daily maintenance (requirement in all species is 40 I.U/kg B.W.).

Control :

1- The minimum daily maintenance requirement must be at least given (40 I.U/kg) daily.

2- In formation of practical diets for all species the daily allowance of vit. A commonly increased but 50-100% of the daily maintenance requirement, particularly during pregnancy, lactation and rapid growth of animals.

3- I/M injection of 3000-6000 I.U/kg B.W., vit. A at intervals of 2 months gives optimum plasma and hepatic levels of vit. A.

N.B:

Ideally the last injection should given 30 days (one month) before parturition for pregnant cattle and buffaloes.

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VITAMIN E AND / OR SELENIUM DEFICIENCY

Synonyms:

Vitamin E- selenium responsive diseases. Or Nutritional muscular dystrophy (NMD). Or.

Enzootic muscular dystrophy.

Etiology:

Primary or secondary deficiency of vitamin E and / or selenium example :

1- Feeding diets which are deficient in vitamin E and / or selenium as feeding inferior quality hay or straw and an root crops.

2- Feeding diets which are incorporated with excessive quantities of polyunsaturated fatty acids (myopathic agent).

Clinical findings:

1- NMD occurs in all farm animals species but most important and most common young rapidly growing calves, lambs and foals.

2- There are two major form or syndrome of NMD known in farm animals:

A- acute enzootic muscular dystrophy:

(myocardial dystrophy)

This form occurs most commonly in young suckling calves, and lambs and occasionally in foals.

1- Affected animals may die suddenly without previous signs.

2- In animals under close observation there is :

a- Sudden onset of dullness.

b- severe respiratory distress, accompanied by a frothy or blood- stained nasal discharge.

C- Lateral recumbency.

d- Increased heart rate (tachycardia) up to 150-200/min. and often irregular.

e- Temp. usually within normal range.

f-Affected animals commonly die within 6-12 hours after onset of signs inspite of therapy.

B- Subacute enzootic muscular dystrophy:

(Skeletal muscular dystrophy)

Form occurs most commonly in older calves (rapidly growing calves) and called WHITE MUSCLE DISEASE, and occur in lambs and called " STIFF LAMB DISEASE".

1- Affected animals may be found in sternal recumbency and unable to stand.

2- In animal which are standing the obvious signs are:

a- Stiffness and trembling of limbs.

b- In calves the gait accompanied by rotating movement of bocks.

c- In lambs the gait is " goose- strepping gait".

d- Palpation of muscles (dorso lumber, gluteal and shoulder) found bilaterally swollen and firm than normal.

e- Knuckling at the fetlock and standing on tip- toe due to relaxation of carpal and metacarpal joint.

f- Dyspnea and abdominal type of respiration may be observed if the diaphragm and intercostal muscles severely affected.

G- Temp. usually within normal range but there may be a transient fever (up 41°C) due to the pyrogenic effect of myoglobinaemia and pain.



Standing on tip-toe in white muscle disease (calf)

Diagnosis:

I) History II) Clinical signs.

III) Lab. Diagnosis:

a-Estimation of muscle specific enzymes:

1- Estimation of plasma creatine phosphokinase activity (CPK) which is a highly specific for cardiac and skeletal muscles degeneration. Normal 25-65 I.U/ litre and in NMD usually up 1000 but commonly as high as (5000-10 , 000 I.U/ litre).

2- Estimation of serum glutamic oxaloatic transaminase activity (SGOT) which is also an indicator of muscle degeneration but not as reliable as CPK because increase SGOT may indicate indicate also liver damage. (Normal less than 100 I.U / litre in calves and lambs and NMD may up to 300-900 in calves and 2000-3000 in lambs).

b- Determination of vitamin E status:

1- The biochemical role of vitamin E is as an antioxidant.

2- Vitamin E occurs in nature as a mixture of Alpha, Beta, Gamma and Deltatocopherol.

3- Determination of tocopherol level in blood and liver provide a good information on vitamin E status of animals.

C-Determination of selenium status

1- The biochemical role of selenium is a component of the enzyme glutathione proxidease (GSH-PX).

2- The blood concentration of selenium is positively related to the activity of the enzyme (GSH-PX) in erythrocytes and vise versa.

Sulfure containing amino acids are a precursors of glutathione which is turn out as a substrate for GSH-PX.

3- Determination of blood conc. Of selenium provide a good information on selenium status of the animals (but difficult and expensive).

D- Glutathione peroxidase: (GSH-PX)

1- There is a direct relationship between the glutathione peroxidase activity of blood and selenium levels of blood and tissues.

2- While blood level of GSH – PX are:

- Deficient if less than 30 mu/mg hemoglobin.
- Marginal if 30-60 mu/mg hemoglobin.
- Adequate of greater than 60 mu/mg hemoglobin.

N.B:

Because selenium is incorporated into erythrocytes GSH – PX only during erythropoisesis, an increase in enzyme activity of blood will not occur for 4-6 weeks following selenium administration.

E- Creatine: creatinine ratio in urine:

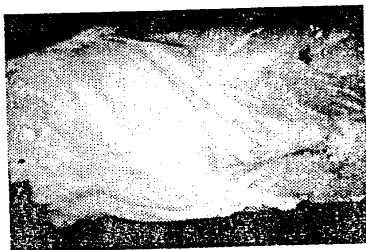
Difficult due to its dependence on collection of 24 hours urine samples.

F- Histopathology:

Hyaline degeneration (coagulation necrosis) observed.



Whitish streaks which appear in the musculature are due to calcification following Zenker's degeneration (Vit E and selenium deficiency in a calf)



Neck muscles of a vitamin E deficient calf (Calcified streaks)

Treatment:

A combined mixture of selenium and alpha tocopherol (potent form of tocopherol) is recommended in treatment of NMD in calves, lambs and foals by single I/M injection of sodium or potassium selenite and alphatocepherol acetate.

Control :

1- give diet or ration adequet in vitamin E and selenium requirement but free of poly unsatturated fatty A.

2- Barium selenate can used and give a slow release which give protection up to 5 months, for the newborn if given to pregnant animals as it can . pass the placental barrier.

DISEASES CAUSED BY DIETARY DEFICIENCY OF CALCIUM, PHOSPHOROUS AND VITAMIN D

(1) Rickets

Definition :

Rickets is a ND disease of young growing animals (calves and lambs) and less commonly in foals) characterized by defective calcification of the growing bones.

The poorly mineralized long bones are subjects to pressure distortions.

Etiology:

Deficiency of one or more of calcium phosphorous and vitamin- D which may be exacerbated by rapid growth rate.

Calcium deficiency

1- Primary calcium deficiency due to lack of calcium in diet seldom occurs.

2-Secondary calcium deficiency due to marginal calcium intake aggravated by a high phosphorous intake is not uncommon.

Phosphorous deficiency

Phosphorous deficiency is usually " primary " under field conditions, and may be exacerbated by a deficiency of vitamin D and possibly by an excess of calcium.

Vitamin – D deficiency

Vitamin –D deficiency usually due to lack of ultraviolet solar irradiation of skin. caused with deficiency of preformed. Vitamin – D complex in diet.

- vitamin D_2 (calciferol) in sun cured hay.

-Vitamin D_3 (cholecalciferol) is produced from its precursor 7-dehydrocholesterol in mammalian skin and by natural irradiation with ultraviolet.

-Vitamin D_4 , D_5 in fish oils.

Pathogenesis :

1- Deficiencies of Ca^{++} ph^{++} and vit. D result in defective mineralization of the osteroid and cartilagionous (calcification) matrix of the developing bones.

2- Poorly calcified spicules of diaphyseal bone and epiphyscal cartilage subjected to normal stresses resulting in bowing of long- bones and broading of the epiphyses with apparent enlargement of the joints.

Clinical findings:

1- Stiffness in gait, lameness and tendency to lie down.

2- Abnormal curvature of long bones, usually forward and out word particularly at knee joint.

3- Enlargement of the limb joints, especially of the fore legs.

4- Enlargement of costochondrol junctions (Rickets Rossetti).

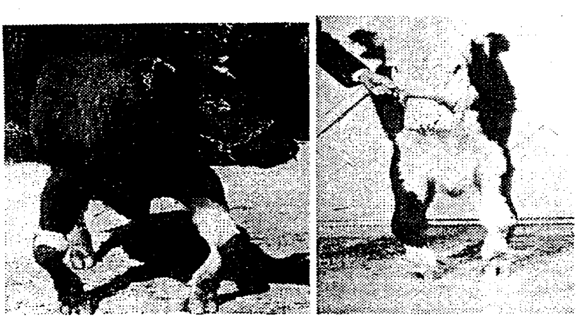
5- Arching of the back.

6- Softness of jaw bones, that severely affected animals unable to close their mouth and tongue protrudes.

7-Dental abnormalities, in the form of delayed and irregular teeth eruption and teeth are poorly calcified with pitting, grooving and pigmentation.

8- Dyspnea and chronic ruminal tympany may resulted from deformity of chest.

9- Hypersensitivity, tetany and recumbency in final severe stages of the disease.



Outward bowing of the forelimbs with enlargement of joints (rickets)



Rickets in a steer before and after vitamin D therapy

Diagnosis:

(I) History (II) Clinical signs.

(III) Lab. Diagnosis: -

A-Estimation of plasma alkaline phosphatase:

Always elevated up to 5.07 units (normal 2.75).

B-Estimation of serum Ca, ph and vit. D:

1- Serum calcium level usually lowered only in the final stages to 8.5 mg% (N = 10-12).

2- Serum phosphorous if the cause, its level will be below 3 mg % (N = 5-7).

3- Serum concentration of 25- hydroxyl vitamin D may be non-detectable.

IV- Radiographic exam. of bones and joints:

Rachitic bones have a characteristic:

1- Lack of density.

2- Ends of long bones have a wooly or motheaten appearance.

3- Long bones have a concave or flat, contour, instead of normal convex.

V- Histopathological exam. Of a small piece of costochondral junction:

Treatment and control :

Lesser deformities recover with suitable treatment but gross deformities usually persist.

Single parenteral I/M injection of 5000 units per lb B.W vitamin D with meet the need of the animals for 3-6 months.

In the same time, animals must receive adequate calcium (from limestone, bone meal, molasses etc.), phosphorous (bran, sodium acid phosphate, disodium phosphate......... etc.)

Exposure of the animal to ultraviolet solar irradiation early morning and early evening.

(II) Osteomalacia

(Milk- lameness, chronic hypophosphataemia)

(Milkleg disease)

Definition:

Osteomalacia is a nutrional deficiency disease of mature animals affecting bones and characterized by " osteoporosis " which manifested clinically by lameness and high susceptability for bone fracture.

Etiology:

The same as for rickets except that predisposing cause is not the increased requirement for growth but the drain of pregnancy and lactation.

Pathogenesis:

1-The drain of pregnancy and lactation caused resorption of bone minerals which leading to osteoporosis and bone weakness.

2-Bone fracture are commonly precipitated by exercise or any abnormal movement.

Clinical findings:

A- in the early stages; the signs are those of phosphorus deficiency including:

1- Licking and chewing of inanimate.

2- Infertility and lower reproductivity.

B-in the latter stages the signs are specific to osteomalacia including:

1-Painful condition of bones and joints manifested in animals by " moderate lameness" which always shifting from leg to leg (milk-lameness commonly occur in heavily milking cows).

2- Crackling sounds while walking.

3- Arched back.

4- Hind legs are most severely affected and the hocks may be rotated inward.

5-Fractures of bones and separation of tendon attachment without apparently precipitating stress.

6-Dystocia when the pelvis bones is affected.

Final weakness lead to permanent recumbency and death from starvation.

Diagnosis:

In general the same as for rickets by:

(I) History (II) Clinical signs.

(III) Lab. Diagnosis.

(IV) Radiographic exam. Of long bones: Decrease density of bone shadow only.

Treatment and control :

The same as for rickets.

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HYPOMAGNESEMIC TETANY OF CALVES

Definition :

Hypomagnesemic tetany of calves is a nutrional deficiency disease with a close similarity to lactation tetany and characterized by hyperasthesia, tetany and convulsions and biochemically by hypomagnesaemia.

Etiology:

1- The basic biochemical changes in calf tetany is hypomagnesaemia which accompanied in many cases by hypocalcaemia.

2- The cause of hypomagnesaemia is the dietary deficiency of magnesium exacerbated by high intake of calcium.

3- Milk, in spite of its low magnesium content is adequate source of the element for very young calves because their absorptive capacity is good.

4- Efficiency of magnesium absorption decreases markedly by:

a- Progression in age of the calf up to about 3 months when max. susceptibility to the disease occur.

b- Reduction in transit time in the intestine and this is may be related to the occurrence of the disease in scouring calves.

N.B:

Hypomagensaemic tetany is calves is often complicated in field cases by the coexistence of other diseases particularly enzootic muscular dystrophy (vitamin – E and / or selenium deficiency).

Incidence, occurrence and predisposing factors:

1- Most incidence in calves 2-4 months of age.

2- In calves fed solely on diet of whole milk.

3- Sometimes in milk replacer fed calves and appear to be related to chronic scours and low Mg content of the replacer.

Clinical findings:

1- The calf apprehensive.

2- Constant movement of ears.

3- Retraction of eyelids.

4- Nystigmus (rotation of eye ball).

5- Champing of jaw.

6- Shaking of the head.

7- Hyperesthesia to touch and external stimuli.

8- Fine muscle tremors.

9-Tetany and convulsions after suddan falling down.

10-Involuntry passage of urine and faeces.

11-a- Pulse accelerated

b- Resp. may cease during convulsive attack.

c- Temp. within normal range but may be elevated.

Diagnosis :

(I) History (II) Clinical signs

(III) Lab. Diagnosis :

1- Estimation of serum Mg level ..... usually below 0.8 mg% (N = 2-2.5 mg%).

2- Estimation of magnesium in bone (particularly ribs and vertebrae) is a reliable confirmatory test at necropsy and Ca: Mg ratio above 90 : 1 is indication of severe magnesium depletion (normal ratio 70 : 1).

N.B:

A field test on urine for hypomagnesaemia is the xylidyl blue test which gives good agreement with sophisticated lab. Tests. The test based on colorimetric estimation of urine magnesium.

Treatment :

1- Mag. Sulphate injections (100 ml 10% solu.)

2- Follow up supplementation of the diet with magnesium oxide or magnesium carbonate:

- 1gm daily mag. Oxide for calves up 5 weeks of age.
- 2gm daily mag. Oxide for calves up 5-10 weeks of age.
- 3gm daily mag. Oxide for calves up 10-15 weeks of age.

3- Chloral hydrate narcosis or tranquilization with ataractic drugs as chloro- promazine Hcl to avoide death from resp. paralysis .

Prevention :

1- Supplementary feeding of magnesium, begin during the first 10 days of life and should continue until at least 15 weeks of age with the same mentioned therapeutic doses.

2- Mag. Bullets two of sheep size per calf.

Trace element:

1- Selenium

2- Copper

3- Iron

4- Cobalt

5- Zinc

6- Manganese

7-Iodine

COPPER DEFICIENCY

Copper deficiency occurs primarily in young ruminants resulting in a range of clinical manifestations including:

- Unthriftiness
- Diarrhea
- Lameness
- Demyelination of CNS in neonates, and
- Falling disease.

Etiology:

Copper deficiency may be primary or secondary

a-primary copper deficiency:

Occur when the copper intake in the diet is inadequate e.g.: feeding plants grown on copper deficient soil.

b-Secondary copper deficiency:

occur when the copper intake in the diet is sufficient but the utilization of copper by tissues is impeded due to dietary excess of Molybdenum and inorganic sulfate alone or in combination.

b-Unthriftiness, anemia, scoring and osteoporosis in extreme deficiency.

N.B:

Molybdenum, interfere with copper absorption from gut mucosa, copper storage in liver, and copper utilization by tissues.

Pathogenesis:

Copper play an important role in " tissue oxidation " by formation of copper- containing enzymes.

e.g.:

- Cytochrome oxidase
- Ceruloplasmin (copper- protein complex).
- Superoxide dismutase.
- Tyrosine oxidase and lysyle oxidase.

Therefore the pathogenesis of most of lesions of copper deficiency attributed to faulty tissue oxidation due to failure of these enzyme system.

A-Skin :

1- Copper deficiency inadequate keratinization which result from imperfect oxidation of the free thiol. Groups.

B- Body weight

Copper deficiency lead in later stages into " retardation in growth" which attributed to impairment of tissue oxidation which interference with intermediary metabolism".

C- Diarrhea:

1- Copper deficiency causing diarrhea due to functional disturbances, as there is no histopathological changes in gut mucosa.

2- Diarrhea is usually only a major clinical finding in secondary copper deficiency associated with molybdenosis.

D-Anemia:

Copper deficiency result in anemia because copper is necessary for reutilization of iron liberated from normal breakdown of hemoglobin for resynthesis of hemoglobin by end of life span.

E-Bone:

Copper deficiency result in " osteoporosis " due to depression of osteoblastic activity.

F- Connective tissue (CT):

1- Copper is a component of the enzyme lysyl oxidease which secreted by cells involved. in the synthesis of elastin component of C.T.
2- Elastin has important function in maintaining the integrity of tissues such as ligaments; tendon etc.

G- Heart :

Copper deficiency leads to myocardial degeneration (falling disease) which may be attributed to either:

1- Interference with tissue oxidation.
2- Terminal manifestation of anemic anoxia.

H- Nervous tissue:

Copper deficiency causing demylination of myelin sheats which may be attributed to anemic anoxia.

I-Immune system:

Copper deficiency causing impaired function of the immune defence system and subsequent increased susceptibility to infection.

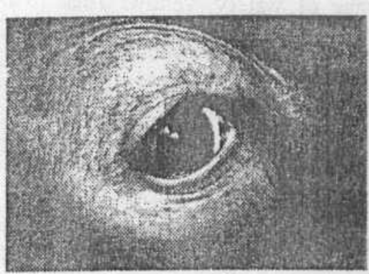
Clinical findings:

Copper deficiency in cattle produces general syndrome and specific diseases:

A- General syndrome:

1- In adult cattle there is unthriftness, loss of milk production and anemia and the coat color is affected, red and black cattle changing to a bleached, rusty red and the coat itself becomes rough and staring.

2- In calves, poor growth, increased tendency for bone fracture and sometimes chronic diarrhea. In some cases ataxia develops after exercise.



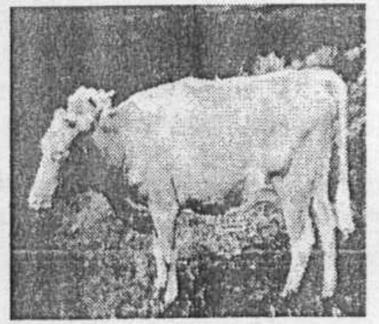
Depigmentation and loss of hair Around the eye (hypocuperosis)



rough hair and depigmentation



Copper deficient cow showing signs of the deficiency as a rough hair coat and patchy areas of lost hair



The same cow after copper therapy.

B-Specific diseases:

a- Falling disease of cattle:

Cows in apparently good health condition throw up their head, bellow and fall. Sudden death after struggle feebly on their sides.

b-Unthriftness (pine) of calves:

progressive unthriftiness, emaciation and grayness of hair especially around the eyes in black cattle.

c-peat scours (teart) of cattle:

1- Persistent diarrhea with passage of watery, yellow green to black faeces with an inoffensive odour.

2- The faeces are released without effort, often without lifting the tail.

3- Severe debilitation result although the appetite remain good.

4-The hair coat is rough and depigmentation is manifested by reddening or gray flecking, especially around the eyes in black cattle.

Diagnosis :

A) History

B) Clinical signs

C) Lab diagnosis

a-Estimation of plasma and liver copper :

1- Plasma copper level bellow 20 ug/dl represent hypocuprosis (N = 20 – 60).

2- Liver copper value below 30 mg/ kg dry matter in cattle and below 80 in sheep considered low (N = above 100 in cattle and above 800 in sheep).

N.B: Estimation of copper content of hair is now acceptable as a diagnostic aid (N 6.6 – 10.4 mg/kg).

B-Estimation of copper containing enzymes: ESOD

station of " erythrocyte superoxide dismutase" value ranges from 2-6 I.U /mg hemoglobin in hypocuprosis.

C-Estimation of copper – protein complex :

(Ceruloplasmin)

1- Ceruloplamsin contains greater than 95% of the courting copper in normal animals.
2- There a highly significant correlation between plasma copper level and plasma ceruloplasmin .
3- Normal plasma ceruloplasmin in cattle 120-200 mg/dl and in sheep 40-100.

N.B:

A high proportion of cows in problems herds may have a Heinz-body anemia without evidence of hemoglobinuria and the severity of anemia will be related to hypocupremia.

Treatment :

1- Oral dosing with 4gm copper sulfate for calves 2-6 months of age and 8-10 gm for mature cattle, given weekly for 3-5 weeks (1/4 dose for sheep).
2- Single parenteral injection of copper glycinate 400 mg for cattle and 150 for sheep by I/M or S/C routes with continue for 2-3 months
3- Supplementation of the diet of the affected animals with copper sulfate added to the mineral salt mixture at level of 3-5 % of the total mixture.

Prevention :

Supplemention of the diet with minimum requirement of copper (10 mg copper /kg matter for cattle and 5 for sheep) by any of several different ways:

1- Oral dosing or dietary supplementation of copper sulfate: 4 gm for cattle and 1.5 gm for sheep weekly.

2- Mineral mixtures of salt licks: Containing 2 % copper – sulfate for cattle (1/4 – ½ %) for sheep.

3- Parenteral injection of copper : Copper glycinate, copper methionate, copper calcium edebate 400 mg for cattle (150 for sheep).

N.B:

For sheep single injection of 45 mg copper glycinate in mid pregnancy is sufficient to prevent sway back in lambs.

4- Copper oxide needles : Fragments of oxidized copper wire up 8 mm in length and ½ mm. Diameter for oral dosing considered one of the most effective and safest methods for control of copper deficiency in ruminants.

The needles retained in fore stomachs and abomasum for 100 days or more and copper slowly released, absorbed and stored in liver.

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ZINC DEFICIENCY

Zinc deficiency commonly occurs in pigs and ruminants (cattle, sheep, goat .. etc.) and characterized clinically by :

- Alopecia.
- Parakeratosis .
- Abnormal hoof growth.
- Lameness.

- Unthrifteness

Etiology:

Primary or secondary zinc deficiency :

1- Primary zinc deficiency occur due to low dietary zinc.

2-Secondary zinc deficiency occur due to factors influence the availability of zinc from soil e.g.: increased fertilization with nitrogen or phosphorus.

Pathogenesis :

1- Zinc is a component of the enzyme carbonic anhydrase which is located in red blood cells and parietal cells of the stomach and is related to the transport of respiratory Co_2 and secretion of gastric Hcl.

2- Zinc associated with RNA function and protein metabolism therefore its deficiency may adversely affect the cell mediated immune system.

3- Zinc related to insulin and glycogen hormones.

4- Zinc has a role in keratinization, therefore, its deficiency lead to failure of keratinization which resulting in " parakeratosis " and loss and failure of growth of hair (due to lesions of the arteriolar walls of dermis).

5- Zinc has a role in calcification therefore bones of zinc- deficient animals revealed abnormal mineralization.

6- Zinc has a role in wound healing.

7- Zinc deficiency result in a decrease feed intake and probably the reason for the depression of growth rate in growing animals.

7-Zinc has a role in sexual development resulting in retarded testicular development in ram lambs and complete cessation of spermatogenesis may attributed to impairment of protein synthesis .

Clinical findings:

1- Parakeratosis and alopecia (may affecting 40 % of the skin area in severe cases) .

2- The lesions are most marked on muzzle, ears, neck, kneefold, backs of hind limbs, vulva, anus and tail – head.

3- Animals stunted growth or below average condition.



Parakeratosis in cattle (zinc deficiency). Skin appears as raw.

Diagnosis :

I) History

II) Clinical signs

(III) Lab. Diagnosis :

A-Estimation of serum zinc – level:

1- Have a good diagnostic value.

2- Normal level 80-120 ug/dl : cattle and sheep.

B-Skin biopsy :

Skin scraping yield negative results but " skin biopsy " will confirm the diagnosis of parakeratosis (marked increase in thickness of all elements of epidermis).

Treatment :

I/M injection of zinc oxide suspended in olive oil result in cure within 2 months.

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Control

1- Feeding on zinc sulphate (2-4gm daily).

2- S/C depots of zinc by injection of zinc oxide.

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IODINE DEFICIENCY
(GOITER)

- The cardinal sign of iodine deficiency is goiter.
- The major clinical manifestation of iodine deficiency is neonatal mortality with alopecia and abnormal enlargement of the thyroid gland.

Etiology :

Iodine deficiency may be primary or secondary

1- Primary iodine deficiency occur due to deficient iodine intake in the diet or in drinking water (this related to geographical circumstances).

2- Secondary iodine deficiency occurs due to :

a- High dietary intake of calcium (decrease intestinal absorption of iodine).

b- High dietary intake of diets containing goitrogenic substances e.g. cabbage, turnips, soyabean, linseed meal etc. Goitrogenic substances are thiocyanate, glucosinate or nitrate (restrict iodine intake).

N.B:

Bacterial contamination of drinking water by sewage is a cause of goitre in humans in countries where hygiene poor.

Pathogenesis :

1- About 80% of the body iodine is stored in the thyroid gland.

2- Iodine deficiency results in a decrease production of thyroxine hormone and subsequently stimulation of the secretion of thyrotrophic hormone by pitutary gland.

3- This result in hyperplasia of thyroid tissue and subsequent enlargement of the thyroid gland.

4- The primary deficiency of thyroxine hormone is responsible for hair abnormalities (alopecia) by absence or diminution in the size of hair follicles.

Clinical sings:

1- Still births and weak, newborn.

2- Partial or complete alopecia.

3- Great enlargement of thyroid G.

N.B:

the gland may pulsate with normal arterial pulse, therefore auscultation of jugular furrow may reveal " thyroid thrill due to increased arterial blood supply of the bland.

4- In adults, loss of libido in bull, and failure to express estrus in the cow and decrease milk production have been suggested as a manifestations of hypothyroidism in cattle.



Goiter in newborn calf (thyroid enlargement)

Diagnosis :

I) History

II) Clinical sings

III) Lab. Diagnosis :

1- Estimation of iodine level in blood and milk.

2- Estimation of PBI in plasma (N = 2.4 – 14 ug%).

Treatment :

1- Treatment must be under taken with care as over dose will cause toxicity (iodism).

2- Specific iodine therapy is recommended for treatment e.g. strong iodine solution (Lugol's solution) 10-20 drops in water daily for 2-3 days.

Control :

The use of " iodized salt " containing at least 0.007% iodine is recommended in all areas known or suspected to be iodine deficient.

DISEASES OF NERVOUS SYSTEM

The Neurologic Evaluation:

An accurate history and thorough physical and neurologic examinations are necessary to evaluate a problem involving the nervous system. An understanding of functional neuroanatomy, neurophysiologic concepts, and mechanism of disease is a requisite for accurate interpretation of clinical findings. From the initial clinical assessment, the problem may be defined as diffuse, multifocal, or focal; symmetrical or asymmetrical; painful or non-painful; progressive, regressive, or static; and mild, moderate, or severe. In addition, the anatomic locations can be determined. The potential mechanisms of disease must also be considered in determining differential diagnoses. Further diagnostic tests include clinicopathologic tests (on serum, blood, urine, feces, and CSF), diagnostic imaging (including plain and contrast radiography and other imaging techniques), and electrodiagnostic testing.

History

Neurologic diseases tend to have a species, age, breed, and occasionally a sexual predilection. The primary complaints for neurologic problems often include behavioral changes, seizures, tremors, cranial nerve deficits, ataxia, and paresis or paralysis of one or more limbs. Information about the onset, course, and duration of the primary complaint can be used to determine the most probable disease mechanisms.

Congenital and familial disorders are most common in purebred animals at birth or within the first few years of life. Inflammatory, metabolic, toxic, and nutritional disorders can occur in any species, breed, or age; tend to have an acute or subacute onset; and are usually progressive. Vascular and traumatic disorders have an acute onset and

rarely progress after 24 hr. Most degenerative and neoplastic disorders tend to occur in older animals (except for familial neuronal degeneration) and have a chronic onset and progressive course. Many idiopathic disorders begin acutely and improve over a short time. Information about similar familial problems, concurrent or recent systemic disease, vaccination status, other affected animals, diet, possible exposure to toxins or trauma, and past neoplastic disorders may be useful to further support certain mechanisms of disease.

Gait Examination:

The gait is observed while the animal walks, trots, gallops, turns, sidesteps, and backs. In large animals, ambulation up and down a grade and while blindfolded may accentuate subtle gait deficits. Evaluation of gait is especially important in ambulatory large animals because postural reactions are difficult to obtain due to size and because spinal reflexes usually are not tested unless the animals are recumbent.

Animals with lesions in the cerebral cortex and diencephalon usually have a relatively normal gait but may circle compulsively. Animals with lesions of the midbrain, pons, and medulla oblongata have paresis or paralysis of the limbs, with deficits often more severe on the side of the lesion. Cerebellar lesions produce ataxia and dysmetria. Vestibular dysfunction causes ipsilateral falling, rolling, or circling. If no abnormalities are found on evaluation of the head, but the gait is abnormal, a lesion most likely is located in the spinal cord, peripheral nerves, or muscles.

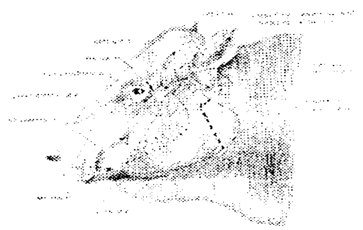
Examination of head and cranial nerves

The mentation, head posture, coordination, and cranial nerve functions are observed during evaluation of the head. Abnormal findings are due to lesions above the level of the foramen magnum in the

cerebrum, the brain stem (diencephalon, midbrain, pons, or medulla oblongata), or the cerebellum. Dementia, compulsive pacing, or other behavioral abnormalities and seizures are frequently due to lesions in the cerebrum or diencephalon. Depression, semicoma, or coma may be due to lesions of the cerebrum, diencephalon, or midbrain. A head turn or compulsive circling without a head tilt is also associated with a cerebral or diencephalic lesion on the side toward which the animal turns. A head tilt is due to vestibular system disease (cranial nerve VIII, rostral medulla oblongata, or cerebellum). Abnormal head coordination, bobbing, and tremors result from cerebellar dysfunction.

The **cranial nerves** are located at specific brain-stem segments; they are simple to test and localize disease to that segment. Abnormal findings are produced by lesions of the peripheral cranial nerve or cranial nerve brain-stem nuclei. If a brain-stem lesion is present, abnormalities are seen in the gait, the thoracic or pelvic limbs, and at times the mental status. If only a peripheral cranial nerve is affected, the other three parts of the examination are normal.





Spinal cervical nerves in bovine head

Cranial nerves in bovine

Special Examination of CNS

Cerebrospinal Fluid Analysis

The analysis of CSF may further aid in determining the mechanism of a CNS disorder (especially inflammation). The technique of collection is simple and safe with practice. Analysis of CSF requires minimal special equipment. Cell counts and identification must be performed within 30 min after collection because cells begin to degenerate after that time. Several techniques are available to concentrate cells so that a differential cell count can be obtained.

CSF is collected from the cerebellomedullary cistern or the subarachnoid space in the lumbar region. An increase in protein is often associated with encephalitis, meningitis, neoplasia, or spinal cord compression. Increased cellular content occurs most frequently with inflammation of the CNS. Neutrophils are indicative of bacterial infections, subarachnoid hemorrhage (RBC are also present), brain abscess or a steroid-responsive suppurative meningoencephalitis, or in some cases, necrosis within a tumor. Increased numbers of lymphocytes, monocytes, and neutrophils are most common in granulomatous meningoencephalitis, fungal infections, toxoplasmosis, and neosporosis. Cultures of CSF may demonstrate the causative agent in bacterial and fungal infections. Paired serum and CSF titers for canine distemper virus, cryptococcus, toxoplasmosis, neosporosis, Rocky Mountain spotted fever, ehrlichiosis, and borreliosis can assist in diagnosis of these infections.

Electroencephalogram (EEG):

An electroencephalogram (EEG) is a recording of the electrical activity of the cerebral cortex, which is influenced by subcortical structures. The EEG is consistently abnormal in hydrocephalus,

meningoencephalitis, head trauma, and cerebral neoplasia. An EEG may determine whether seizure discharges are focal or diffuse.

Modes of nervous dysfunction:

1-Excitation or irritation signs

Increased activity of the organ occurs because of an increase in the number of nerve impulses received either because of excitation of neurons or by facilitation of the passage of stimuli. The excitability of nerve cell can be increased by many factors including stimulant drugs. The major manifestation of irritation of nervous tissue are convulsion and muscle tremor in locomotor system and hyperaesthesia and paraesthesia in the sensory system

2-Release signs

Exaggeration of normal nervous system activity occurs when lower nervous centers are released from the inhibitory effects of higher centers. Cerebellar ataxia is a classical example. In the absence of cerebellar control, combined limb movements are exaggerated in all mechanisms of actions including rate, range, force and direction

3-Paralysis due to tissue destruction.

Different degrees of depression of activity can occur with nerve cells. The terminal stage being complete paralysis when nervous tissue is destroyed. The depression of activity may result from failure of supply of oxygen and other essential nutrients either directly from their general absence or indirectly because of failure of local circulation. Infection of the nerve cell itself may cause initial excitation, then depression of function and finally complete paralysis when the nerve cell dies. Signs of paralysis are constant and manifested by muscular paresis when the motor system is affected and by hypoaesthesia or anaesthesia when the sensory system is involved.

4-Nervous shock

An acute lesion of the nervous system causes damage to the nerve of the lesion in addition temporary cessation of function in parts of the nervous system not directly affected the loss of function in these areas is temporary and usually persists for only a few hours. Stunning is the obvious example.

Mechanisms of Nervous Disease:

Most disease processes affecting the nervous system are congenital or familial, infectious or inflammatory, toxic, metabolic, nutritional, traumatic, vascular, degenerative, neoplastic, or idiopathic.

Congenital disorders may be obvious at birth or shortly after (eg, an enlarged head from hydrocephalus or an incoordinated gait from an underdeveloped cerebellum). Some familial disorders (eg, lysosomal storage diseases) cause a progressive degeneration of neurons in the first year of life, while others (eg, inherited epilepsy) may not manifest for 2-3 yr.

Infections of the nervous system are due to specific viruses, fungi, protozoa, bacteria, rickettsia, and algae. Nonspecific inflammations such as steroid-responsive meningoencephalomyelitis and granulomatous meningoencephalomyelitis may be immune-mediated.

Toxicity of the nervous system is most frequently caused by organophosphates, pyrethrins, carbamates, bromethalin, metaldehyde, ethylene glycol, metronidazole, theobromines, and sedatives. Botulinum, tetanus, and tick toxins, as well as coral and tiger snake venom intoxication, cause neurologic signs.

Metabolic alterations of nervous system function most commonly result from hypoglycemia, hypoxia or anoxia, hepatic dysfunction, hypocalcemia, hypomagnesemia, hypernatremia, hypokalemia, and

uremia. Hypothyroidism, hyperthyroidism, hypoadrenocorticism, and hyperadrenocorticism are endocrine disorders that can cause neurologic dysfunction.

Thiamine deficiency results in ataxia, stupor, and coma or seizures in cattle. Deficiency of vitamin B_6 may cause seizures.

Trauma to the PNS and CNS cause focal and multifocal neurologic signs from physical damage, hemorrhage, edema, and progressive formation of free radicals and nervous system destruction that is usually complete in 24-48 hr.

MANIFESTATIONS OF DISEASES OF THE NERVOUS SYSTEM

I- Mental state:

The excitation state includes mania and frenzy. Both are manifestation of general excitation of the cerebral cortex:

A- Mania:

The animal acts in a bizarre (strange) way and appear to be away of the surroundings. Maniacal actions include licking, chewing of foreign materials, abnormal voice, constant bellowing, apparent blindness, walking into strange surroundings, drunken gait and aggressiveness in normal docile animals.

Diseases causing mania include:

a- encephalitis

b- degenerative lesions of the brain (polioencephalomalacia)

c- Toxic and metabolic diseases of the brain: such as nervous form of acetonemia, acute lead poisoning, poisoning with carbontetrachloride, and hepatic insufficiency.

B- Frenzy:

Frenzy is characterized by violent activity with little regards to the surroundings. The animal's movement is uncontrolled and dangerous to other animals in the group or to the human attendants. It is often accompanied by aggressive physical attacks.

Diseases of nervous system causing frenzy include:

a- Encephalomyelitis

b- Toxic and metabolic diseases of the brain: such as hypomagnesemic tetany.

c- Depression of mental state

It includes somnolence, lassitude, narcolepsy/catalepsy, syncope and coma. They are manifestations of depression of cerebral cortex

The depression leading to coma may result from:

a- Encephalomyelitis and encephalamalacia.

b- Toxic and metabolic diseases of the brain such as uremia, hypoglycemia, toxemia, septicemia and hepatic insufficiency.

c- Heat stroke.

d- Hypoxia of the brain caused by peripheral circulatory failure in milk fever.

The depression leading to syncope (fainting) may result from:

a- Acute heart failure leading to acute cerebral anoxia

b- Spontaneous cerebral hemorrhage.

c- Lightning stroke and electrocution.

II- Involuntary movements:

1- Tremors:

It is continuous repetitive twitching of the skeletal muscle, which is visible and palpable. The muscle unit involved may be small leading to movement of local skin area and this is called fasciculation. When the

muscle unit involved is extensive, the movement may be coarser and sufficient to move the extremities or parts of the trunk.

Tremors may be caused by:

a- diffuse diseases of the cerebrum, cerebellum and spinal cord.

b- Degenerative nervous system diseases: hypomyelinogenesis in newborn calves.

c- Toxic and metabolic diseases of nervous system: early stage of hypocalcemia (fasciculation of the eyelid and ears)

2- Tetany:

It is a continuous contraction of muscles without tremors usually associated with clostridium teteni intoxication following localized infection with the organism

3- Convulsions

these are violent muscular contraction affecting parts or all parts of the body occurs for short period but repetitive in some cases as encephalitis. They begins and end abruptly. Convulsions result from abnormal electrical discharges in forebrain neurons that reaches the visceral and somatic motors initiating spontaneous involuntary paroxysmal movements.

The causes of convulsion may be:

a- Intracranial convulsions

1- Encephalomyelitis and meningitis.

2- Encephalomalacia.

3- Acute brain edema.

4- Brain ischemia.

5- Local lesions caused by trauma, abscess, tumor, parasites or hemorrhage.

b- Extracranial convulsions:

1- Brain hypoxia in acute heart failure.
2- Toxic and metabolic diseases of the nervous system including:
   a. Hepatic insufficiency
   b. Hypoglycemia
   c. Hypomagnesemia
   d. Inorganic poisons such as chlorinated hydrocarbons
   e. Clostridium *spp*.intoxication.

DISEASES OF THE NERVOUS SYSTEM
HEAT STROKE (HYPERTHERMIA)

Definition

It is the clinical syndrome of elevation of body temperature due to excessive heat production or absorption or deficient in heat loss

Etiology

1- High environmental temperature
2- Prolonged severe muscular exertion especially when
   a. the humidity is high
   b. the animal is obese
   c. the animal is confined with inadequate ventilation
3- Neurogenic hyperthermia: due to damage to hypothalamus as a result of spontaneous hemorrhage.

Pathogenesis

Heat stroke leads to hyperthermia, which leads to:

1- Increase the metabolic rate by 40-50% and the liver glycogen stores are rapidly depleted and extra energy is produced from endogenous metabolism of protein loss of body weight and lack of muscle strength
2- Dryness of the mouth leading to thirst (increased water intake)

3- Increases heart rate due to peripheral vasodilatation
4- Increased respiratory rate due to the direct effect of heat on respiratory centers.
5- Decreased urine secretion because of the reduction of renal blood flow resulting from peripheral vasodilatation
6- Depression of the CNS when temp. reaches critical level (42.5 °C).

Clinical signs

1- Increase body temp. more than 42 °C which is different from fever (seldom exceeds 41 °C)
2- Increased heart and respiratory rates.
3- Initial salivation followed soon by dryness of muzzle and absence of sweating.
4- The animal seeks cold places to splash (wet or spray) itself
5- Nervous signs include restlessness initially but soon the animal becomes dull and stumbles while walking and tend to lie down.
6- In severe heat stroke, there is convulsions, terminal coma and death.

Diagnosis:

I- History: severe environmental temperature
II- Clinical signs
III- Differential diagnosis from fever caused by infectious factors.
IV- Post Mortem changes:
  a. Early start of rigor mortis
  b. Peripheral vasodilatation

Treatment

1- Cold applications such as immersion, spraying or rectal enema
2- IV administration of fluids such as saline

3- Provide adequate amount of drinking water with shade and air movement

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ENCEPHALOMALACIA

Definition

Degenerative disease of the brain, using softening of the white matter (leukoencephalomalacia) . or the grey matter (polioencephalamalacia) producing initial irritation signs followed by paralysis.

Etiology

1- Hepatic encephalopathy due to high level of ammonia
2- Thiamin deficiency
3- lysosomal storage disease
4- Prolonged parturition of calves
5- Plant poisoning

Clinical signs:

1- in early stage, there are irritation signs such as
  a. Muscle tremors,
  b. Opisthotonus, nystigmus and convulsions.
2- **Late stage:**
  a. Paralysis of varying degrees, accompanied by dullness, somnolence,
  b. Blindness, ataxia, circling and terminal coma

Diagnosis

I- History
II- Clinical signs

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Treatment

The disease is irreversible so the animal can be maintained for survival on supportive treatment so that it can be fattened by slaughter

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MENINGITIS

Definition:

Inflammation of the meninges that is mostly caused by bacterial infection and characterized clinically by fever, cutaneous hyperesthesia and rigidity of the muscles.

Meninges are 3 layers of connective tissue around the brain and spinal cord: dura matter, pia and arachnoid membranes. The CSF presents between the pia and arachnoid membrane to provide nutrient and absorbs the ipacts of shock.

Etiology

1- Adult cattle

I- Viral diseases

a. Malignant catarrhal fever

b. Sporadic bovine encephalomyelitis

II- Bacterial diseases

a. Listeriosis

b. Hemophilus somnus

c. Tuberculosis

d. Leptospirosis

2- Young animals

a- streptococcal and coliform septicemia

b- hematogenous extension from omphalophlebitis

Pathogenesis

1- Inflammation of meninges causes local swelling and interference with the blood supply to brain and spinal cord.
2- The signs produced by meningitis are due to irritation of both central or peripheral nervous system
3- In spinal meningitis, there is muscular spasm with rigidity of limbs and neck, arching of back and skin hyperesthesia.
4- In central (cerebral) meningitis, muscle tremors and convulsions
5- Pyrogenic bacterial infection leads to exudation into the CSF leads to signs of increased intracranial pressure.
6- Since the cause is mostly bacterial, there toxemia and fever

Clinical signs

1- Fever and toxemia
2- Nervous signs include
   a. Trismus (contraction of muscles of mastication and opisthotonus (contraction of muscles of back)
   b. Rigidity in the neck and back
   c. Muscle tremors
   d. Cutaneous hyperesthesia as light touch of the skin produces severe pain.
   e. Disturbance in consciousness:
      i. Early stage: there is excitement or mania.
      ii. Late stage: there is a drowsiness and eventual coma.
   f. Blindness (moving into objects that the animal should avoid) is common with cerebral meningitis

Diagnosis

1- History

II- Clinical signs: particularly skin hyperesthesia, blindness and muscle tremors

III- Lab examination of CSF to confirm bacterial infection

IV- Hemogram: leucocytosis

Treatment

I- Treatment is directed mainly to overcome the bacterial infection

1- The use of parental antibiotic selected by culture and sensitivity
2- Chloramphenicol has been used because it can pass the blood-brain barrier but prohibited in food-producing animals.
3- Trimethoprim-sulphonamide combination with or without gentamycin which has a synergistic effect with the combination has shown to be effective in meningitis.
4- Penicillin and oxytetracycline are effective for treatment of meningitis in cattle due to hemophilus somnus when the treatment begins early

II- Supportive treatment: by intravenous or stomach tube feeding

III- Tranquilizer in early stage of excitation or mania and nervous stimulant in late stage of drowsiness and coma



Structure of brain

MENINGOENCEPHALITIS (ME)

Definition

Inflammation of the meninges (meningitis) and inflammation of the brain (encephalitis) often occur simultaneously (meningoencephalitis) in the same animal, although either can occur separately. In animals with meningoencephalitis, the clinical signs of meningitis often precede the clinical signs of encephalitis and may remain the predominant feature of the illness.

Etiology and Pathogenesis:

The incidence of meningitis and encephalitis is fairly low compared with that of infections of other organs. This appears to result from the better protection offered to the nervous system by its barriers, rather than to a scarcity of infectious agents that can attack the nervous system.

1- Infections of the nervous system often are the result of some injury to its protective barriers. In all species, direct extension of bacterial or mycotic infections to the CNS can occur from sinusitis, otitis media or interna, or vertebral osteomyelitis, these infections can also be secondary to deep bite wounds or traumatic injuries adjacent to the head or spine.
2- Iatrogenic infections are possible from contaminated spinal needles or surgical instruments. Infections may occur if CSF taps are performed in animals with bacteremia.
3- Brain abscesses also can arise from direct infections or by septic embolism of cerebral vessels. In chronic brain abscesses, an adjacent or occasionally diffuse fibrinous leptomeningitis may develop.

4- A spontaneous bacterial meningitis or meningoencephalitis can develop (although less commonly than in farm animals) by various bacteria (Pasteurella multocida, Staphylococcus aureus , S epidermidis, S albus and Actinomyces spp) have been isolated.
5- Bacterial meningoencephalitis often affects neonatal farm animals as a sequela of septicemia caused by Escherichia coli
6- In older or adult animals, well-recognized disease entities, such as thrombotic meningoencephalitis (TME) of cattle (*Haemophilus somnus*) also cause meningoencephalitis by the hematogenous route.
7- Listeriosis, which is caused by *Listeria monocytogenes* and is a common infection in cattle, sheep, and goats, is an example of a multifocal brain-stem meningoencephalitis that ascends to the CNS via transaxonal migration in cranial nerves.
8- *Pasteurella haemolytica* and *P multocida* , although usually resulting in fibrinous pneumonia and hemorrhagic septicemia in ruminants, occasionally produce a localized fibrinopurulent leptomeningitis.

8- Viral diseases: malignant catarrhal fever and sporadic bovine encephalomyelitis.

Clinical Findings and Lesions:

The usual signs of meningitis are

1- Fever, hyperesthesia, neck rigidity, and painful paraspinal muscle spasms.
2- In diffuse meningoencephalitis due to any agent, depression, blindness, progressive paresis, cerebellar or vestibular ataxia, opisthotonos, cranial nerve deficits, seizures, dementia, agitation, and depressed consciousness (including coma) can develop.

depending on the rapidity of onset, severity of pathology, and location of the lesions.

3- In neonatal infections, omphalophlebitis, polyarthritis, and ophthalmitis with hypopyon can accompany the CNS inflammation.

4- Because of its unusual pathogenesis, listeriosis often causes asymmetric vestibular dysfunction, with head tilts and circling. This is in addition to other cranial nerve deficits, such as facial and pharyngeal paralysis.

5- In ME of cattle, the nervous signs tend to be peracute, with sudden collapse and profound depression of consciousness (stupor or coma). Fever and limb stiffness may be the only signs detectable in the prodromal stages of ME.

Clinical signs of pyogranulomatous meningoencephalomyelitis include

1- neck rigidity, kyphosis, inability to raise the head, reluctance to move (eggshell gait), and limb incoordination (ataxia). Sometimes, bradycardia, vomiting, and

2- in chronic cases, atrophy of cervical muscles may be seen. Cranial nerve signs may include Horner's syndrome and paralysis of any cranial nerve but most commonly the trigeminal and facial nerves.

Pathological changes

The pathological changes characteristic of bacterial meningoencephalitis include

1- Diffuse infiltration of both neutrophils and mononuclear cells into the leptomeninges.

2- Frequently, the entire subarachnoid space of the brain and spinal cord is inflamed.

3- Vasculitis of meningeal vessels and CNS arterioles is often pronounced.
4- Bacteria may also invade the CNS parenchyma, resulting in mononuclear and polymorphonuclear infiltration with large areas of perivascular cuffing.
5- Necrosis and malacia of the CNS may be seen, with infiltrations of macrophages, neutrophils, and plasma cells.
6- Listeriosis uniquely causes microabscesses deep within the CNS parenchyma, which consist of accumulations of neutrophils and microglial cell reaction with central liquefaction necrosis.

Diagnosis:

1- Case history
2- Clinical signs
3- CSF analysis:
   a- The analysis of CSF is the most reliable and accurate means of identifying meningitis or meningoencephalitis.
   b- CSF should be collected whenever history or species or breed predisposition suggests meningitis or encephalitis, or whenever clinical signs suggest a disseminated or inflammatory CNS disorder.
   c- The protein content of the CSF is usually significantly increased (100-5000 mg/dL), with an increase in the globulin component of CSF.
   d- Occasionally, bacteria are seen on cytologic examination of the CSF and identified with Gram's stain. Successful culture of bacteria from CSF is more likely in large animals than in dogs. In some cases, serial blood cultures are more successful for isolation of the causative organism.

e- Viral infections and listeriosis typically produce a mononuclear pleocytosis in CSF; the total cell count and protein levels are mildly to moderately increased, again with an increase in CSF globulin.

f- Granulomatous inflammations usually induce moderate to high cell numbers and increased protein in the CSF. The cell population is predominately mononuclear, and it can be difficult to distinguish between a granulomatous infection (eg, a fungal infection) and idiopathic GME. However, in pyogranulomatous meningoencephalomyelitis, CSF analysis usually reveals a neutrophilic pleocytosis (500-1000 WBC/µL). Cryptococci and occasionally protozoa have been identified in CSF, but usually serology is necessary to confirm mycotic and protozoal infections in vivo.

Treatment:

1- Broad-spectrum antibacterials such as ampicillin, chloramphenicol, tetracyclines, trimethoprim-sulfas, and third-generation cephalosporins are used, but higher than normal dosages may be necessary to achieve and maintain adequate concentrations in the CNS. In farm animals, selection of drugs must be based not only on drug efficacy but also on whether the available drug is appropriate for use in a food animal.

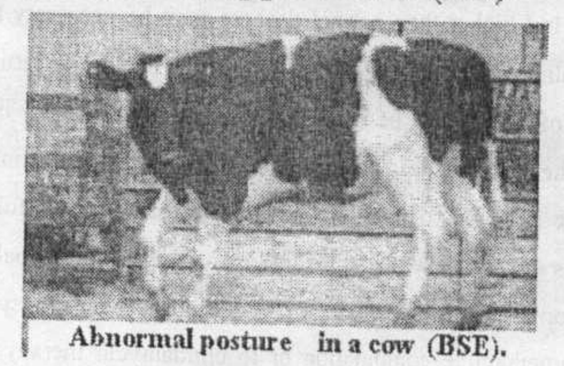
2-Mycotic infections of the CNS have been treated successfully in man, but results in veterinary medicine are less promising. Protozoal infections (eg, toxoplasmosis, neosporosis, sarcocystosis) may respond to a sulfa/pyrimethamine combination or to clindamycin therapy. However, relapse may occur due to the inability to clear encysted organisms from the CNS.

3-Glucocorticoids are usually contraindicated in animals with meningitis or meningoencephalitis with an infectious etiology; however, a high-dose, short-term course of dexamethasone or methylprednisolone may control life-threatening complications such as acute cerebral edema and impending brain herniation.

4- Supportive care should be specific for the needs of the individual animal and may include analgesics, anticonvulsants, fluids, nutritional supplementation, and physical therapy.



High -stepping gait in a cow (BSE).

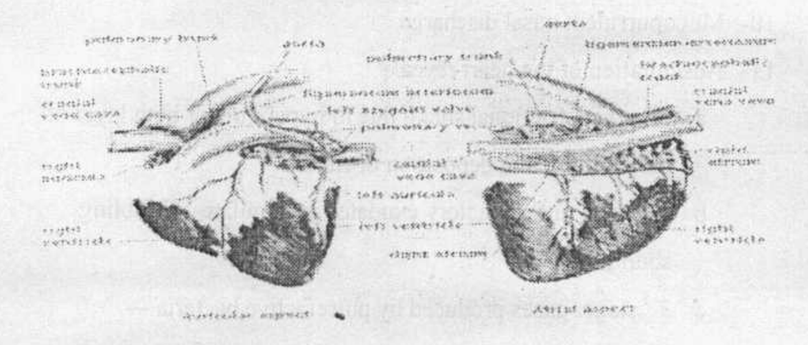


Abnormal posture in a cow (BSE).

DISEASES OF CARDIOVASCULAR SYSTEM

Introduction about the cardiovascular system

The cardiovascular system comprises the heart (the pump), the veins (the vessels leading into the heart), and the arteries (the vessels leading away from the heart). The cusps of the atrioventricular valves keep blood flowing in one direction through the heart, and valves in large veins keep blood flowing in one direction through them as well. The rate and force of contraction of the heart and the degree of constriction or dilatation of blood vessels are determined by the autonomic nervous system and hormones produced either at the heart and blood vessels (i.e., paracrine or autocrine) or at a distance from the heart and blood vessels (i.e., endocrine) Slightly >10% of all domestic animals examined by a veterinarian have some form of cardiovascular disease. Unlike diseases of many other organ systems, cardiovascular diseases generally do not resolve but almost always become more and more limiting and may lead to death. In addition, cardiovascular diseases may be more difficult to detect and quantify because the heart cannot be seen and is protected so well by the rib cage. Therefore, evaluation of the heart depends on heart sounds and murmurs, pressure pulses and the apex beat, the electrocardiogram, and radiology and echocardiology.



TRAUMATIC PERICARDITIS

Definition:

Inflammation of pericardial sac due to penetration by sharp penetrating foreign bodies characterized clinically by recumbence, fever, anorexia, and sharp decrease in milk production

Etiology:

1- Penetration of pericardium by sharp FB.

2- As a sequel to traumatic reticulitis or traumatic reticulopertonitis.

Symptoms:

1- Fever, anorexia, increase pulse and resp. rate.

2- Sharp decrease in milk production

3- Abduction of the fore limbs with arched bach and tense abdomen

4- Animal move and lie down carefully.

5- Animal prefers to stand, except at late stage of disease.

6- Jugular vein pulsation and cord like appearance.

7- Increase venous pressure indicated by resistance to iject drugs.

8- Congested conjunctival blood capillaries due to fever and venous retention.

9- Recumbency

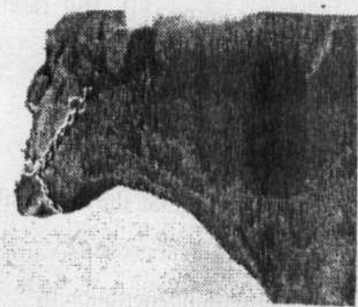
10- Mucopurrulent nasal discharge

11- Auscultation of the heart reveals:

   a. 1st stage: frictional sound due to hyperemia of both layer of pericardium and deposition of fibrin
   b. 2nd stage: inflammatory exudates accumulate—dribbling sound.
   c. 3rd stage: gases produced by putrefactive bacteria --- splashing sound
   d. excessive increase of infl. Exudates – muffling sound

12- Increase area of dullness by percussion.

13- Decrease area of pulmonary resonant sound.



Distension of jugular vein (traumatic pericarditis)



Sternal edema (traumatic pericarditis)

Diagnosis:

1- history

2- clinical signs

3- pain test if animal is standing:

a. Pinching of wither.

b. Percussion on sternum till reaching the reticulum (xiphoid cartilage).

c. Going up and down a slope (groaning down the hill).

d. Stick test: elevation of the animal abdomen using a stick and release suddenly.

Reaction of the animal (positive response):

- Deep rep. movement in buffaloes
- grunting sounds and grinding of teeth in cows

e- Turning to close left side circle – difficult

4- Lab. Examination;

a. WBCs count 15000-30000

b. Neutrophilia: 60-70% (N 30-35 %)

Treatment

1- Surgical interference: rumenotomy then remove FB. But rarely successful

2- Preservative treatment: in pregnant animal till parturition by:

a. Immobilization.

b. Keep the animal with the fore limb raised than the hind limb.

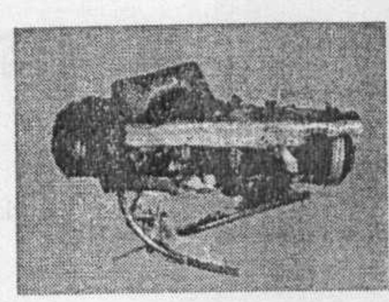
c. Decrease food intake to decrease abdominal pressure

d. Control of infection that may enter with ppenetration by:

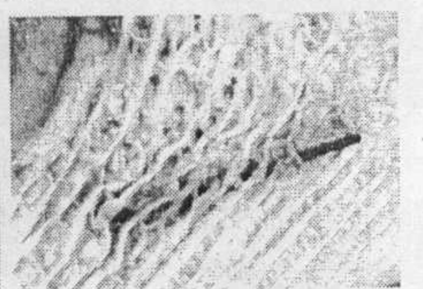
i. Sulphonamides: 2-3 gm orally for 3-4 days

ii. Oxytrtracycline: 2-3 gm dissolved in normal saline and injected IP.

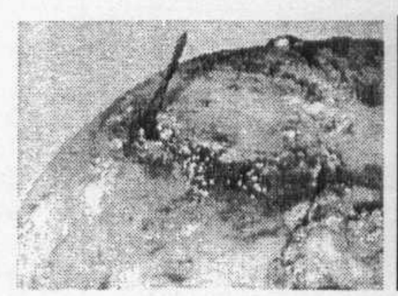
iii. Penicillin-streptomycine combination dissolved in saline and injected IP.



Metal door spring removed from reticulum



A nail is embedded in the mucosa of a cow's the reticulum



A nail has penetrated the reticulum, causing traumatic reticuloperitonitis (hardware disease) and the death of this cow



A piece of wire has penetrated the reticulum and diaphragm before lodging in the pericardium. Pericardial effusion and fibrin deposition resulted from this traumatic injury

Failure of circulatory system includes:

1- Acute heart failure

2- Chronic heart failure

3- Peripheral circulatory failure

ACUTE HEART FAILURE

Definition

Acute cardiac disorder with sudden stop of cardiac output to meet body needs, characterized clinically by:

- Sudden loss of consciousness.
- Falling with or without convulsions.
- Severe paleness of mucosae (m.m).
- Either death or complete recovery from the episode.

Etiology

A- Severe defect in filling (disorder of filling)

B- Failure of the heart as a pump either due to severe tachycardia or bradycardia.

C- Sudden increase in work-load.

(A) Disorder of filling

Due to pericardial tamponade (compression of heart for instance by haemopericardium)

(B) Failure of heart as a pump

May be either due to tachycardia or bradycardia

a-Tachycardia:

1-Myocarditis e.g. bacterial, viral or parasitic (e.g. encephalomyocarditis v.)

2-Nutritional deficiency myopathy e.g. Copper and selenium deficiency

3- Electric strike.

B-Bradycardia

1-Dosing by rapid I/V calcium preparation

2- Sudden increase in work-load due to:

1-Acute anaphylaxis shock

2- Rupture of aortic valve..

Pathogenesis

1- In disorder of filing due to pericardial tamponade and in tachycardia due to shorting of diastolic period, the filling of the ventricles is impossible, therefore cardiac output grossly reduced,

2- In severe bradycardia, the cardiac output also grossly reduced due to excessive decrease in heart rate.

3- In sudden increase in workload cardiac fibrillation occurs, leads to incoordinated contraction, thus no blood ejected from the heart.

4- In all of these circumstances there is a fall in minute volume of the heart which leads to severe degree of tissue anoxia.

5-The brain is the most sensitive organ first affected therefore clinical signs are principally nervous type

6-Pallor occur due to reduction in arterial blood flow

Clinical Findings:

1- Sudden loss of consciousness due to cerebral anoxia.

2- Staggering and falling with or without clonic convulsions.

3- Marked pallor mucosae (m.m)

4- Death usually follows within seconds or minutes and usually accompanied by deep, asphyxia gasps.

5- If there is time for exam, there is absence of pulse and there is either tachycardia bradycardia or absence of heart sounds.

6- In less acute cases the course may be as long as 12-24 hours, and dyspnea and pulmonary oedema are prominent signs.

Diagnosis

I) History

II) clinical signs.

N.B

Insufficient time is available to conduct lab. Diagnosis.

Treatment

Treatment is not usually possible because of the short course of the disease, but the following may be useful.

1- Direct cardiac massage or electrical stimulation but these techniques are restricted to the more sophisticated surgical units.

2- Intracardiac injection of very small doses of adrenaline and as much harm as good.

CONGESTIVE HEART FAILURE

Definition

It is a cardiac disorder in which the heart is unable to maintain circulatory equilibrium and characterized by "congestion of the venous circuit" accompanied by:-

1- Enlargement of the heart and increase in heart rate.

2- Dilation of veins and edema of lungs or periphery.

Etiology

Diseases of the endocardium, myocardium and pericardium

1-Interfere with flow of blood into heart or away from the heart.

2-Diseases which impede the heart action

(A) Valvular diseases

1- Aortic and pulmonary valve stenosis or insufficiency.

2-Rupture of valve or valve chordae

(B) Myocardial diseases

1 -Myocardial asthenia (weakness in myocardium)

2-Myocardial arrhythmia (regulatory in rhythmic control)

3- Myocardial degeneration: toxic or nutritional or neoplastic

e.g.

1 -Myocarditis by bacterial, viral or parasitic.

2- Myocordial dystrophy in copper, selenium and vit. E deficiency.

3- Myocardial neoplasms.

(C) pericardial disease

Hydropericardium, cardiac tamponade and pericarditis.

Pathogenesis

1- Increased heart load to eject blood and weakness of the heart →muscles stimulate the compensatory mechanisms such as increasing heart rate, dilatation and hypertrophy of heart to maintain circulatory equilibrium.
2- At rest. this compensatory mechanism may be able to overcome the circulatory emergency.
3- If more work load is required, as in excitement and exercise, the heart fail to maintain the circulatory equilibrium and congestive

heart failure develop where venous engorgement with blood occur followed by edema.

4- Right side CHF leads to generalized edema whereas left side leads to edema restricted to lungs.

5- Right side CHF may affect liver and kidney:

a. In kidney:

i. Reduced urine flow due to decreased glumerular filtration

ii. Anoxic damage of glomeruli leads to increased permeability to plasma proteins

b. In liver: hepatic dysfunction and disturbance in digestion occurs due to venous congestion of portal circulation.

Clinical signs

* Generally, in very early stages of C.H.F. there is respiratory distress on light exertion and time required for return to normal respiration and pulse rates is prolonged.

* C.H.F. may be referable to failure of left side of the heart. or right side of the heart.

A) Left sided C.H.F. (Signs of lung oedema)

Characterized by

1- Increase rate and depth of respiration at rest.

2- Moist cough, and mucoid bilateral nasal discharge plus frothing from mouth.

3- Moist rales at base of the lungs.

4- Increased dullness on percussion of ventral border of lungs.

5- Terminally. Severe dyspena and cyanosis.

6- Tachycardia and there may be a murmur (referable to left atrioventricular (mitral) or arotic semilunar valves.

7- Epistaxis in severe cases.

8- Corneal opacity (glucoma).

(B) Right sided C.H.F (Body oedema)

1- Oedema (anasarca, ascitis, hydrothorax and hydropericardium).

2- Anasarca charactestically limited to the ventral surface of the body, the neck and the jaw.

3- Tachycardia and dilation of superficial veins (particulary J.V.)

4- Faeces normal at first, but in the late stage profuse diarrhea may be present.

5- Oliguria and albuminuria.

6- Liver severely enlarged and protruding beyond the right costal arch in severe right sided C.H.F.

Diagnosis:

I- History.

II- Clinical signs (observation, palp., percussion, Auscaltation).

III- Venepuncture test.

Increased pressure of blood from needle on venepuncture much greater than normal due to increase venous pressure.

IV- Radiography.

V- ECG.

VI- Echocardiography.

Treatment:

Non-specific treatment is applicable in most cases irrespective of the cause. It consists of:

1- Restriction of activity (to reduce the demand on cardiac output).

2-Diuretic medication (to overcome the load of oedema) orally or I.M.

3- Digitalis glycosides (to improve myocardial contractility) but of little effect in ruminant.

4-Venesection can be used as an emergency treatment in acute pulmonary oedema (4-8 ml of blood / kg B.W may be withdrawn).
5- Paracentesis for drainage of serous cavities.

PERIPHERAL CIRCULATORY FAILURE

Definition:

It is a circulatory disorder characterized by reduction in cardiac output due to failure in the "Venous return ".

Etiology:

Failure in the venous return may be:

1- Vasogenic failure 2- Haematogenic failure

(A) Vasogenic failure

1-Occur when there's peripheral "Vasodilatation" and pooling of blood in B. vessels.
2-This is principally occurring in "Shock" when the blood collected in the dilated splanchic B. vessles.

(B) Haematogenic failure (Hypovolemic failure)

1- Occurs when there is reduction in the circulating blood volume
2- This occurs principally in:
a) Hemorrhage b) Dehydration

Clinical findings:

Failure of venous return, lead to reduction in cardiac output which in turn lead to decrease blood flow to tissues resulting in tissue hypoxia or anoxia, which in turn responsible for the following clinical manifestations:

1- Anorexia, which may by accompanied by thirst.
2- Shallow rapid respiration.

3- Tachycardia accompanied by weak intensity of heart sounds, as well as, abnormalities of pulse amplitude.

4-Low arterial blood pressure (measured either directly by arterial puncture or by indirect methods using sphigmography).

5-Nervous signs include depression, weakness and restlessness, as well as, coma in the terminal stages.

N.B.:

Clonic convulsions may occur but they are not a prominent dart of the syndrome.

6- Cold skin and extremities.

7- Pale m.m with prolonged capillary filling time.

8- Subnormal temperature.

Diagnosis:

I- History

II- Clinical signs

III- Lab. Diagnosis.

1- PCV, Hb, and total erythrocytic count, usually below normal due to haemorrhagic anemia and dehydration.

2- Eosinopenia, lymphocytopenia and thrombocytopenia.

3- Hyperkalaemia.

4- Commonly with P.C.F there's disturbance in the acid base status with metabolic acidosis and lactic acidosis.

Treatment:

-Regardless of the cause, treatment is to restore the circulating blood volume to normal to avoid tissue anoxia.

(a) In Vasogenic Failure

Plasma transfusion is required to overcome shock.

(b) Haematogenic Failure

1- Give whole blood transfusion to overcome hemorrhage.

2- Give isotonic fluid replacement overcome dehydration.

N.B

1- In vasogenic failure, a large dose of corticosteroids (2-3 mg / kg I/V) may be useful as an anticardiogenic shock.

2-In both vasogenic failure and haematogenic failure, a specific bicarbonate (Na bicarbonate 1% I/V) should included to overcome the disturbance in acid-base status i.e marked blood acidosis and lactic acidosis.

ENDOCARDITIS

Definition:

Inflammation of endocardium that may interfere with ejection of blood from the heart by causing insufficiency and stenosis producing congestive heart failure and characterized clinically by murmurs.

Etiology

1- Mostly bacterial infection, which is mostly hematogenous: due to metritis, mastitis,..etc.
2- Bacterial infection include:
    a. Streptococci
    b. Corynebacterium pyogene
    c. Clostridium chauvoei (Blackleg)
    d. Mycoplasma mycoides.

Clinical findings

1- usually a history of ill thrift and periodic reduction in milk production is present
2- Moderate elevated temp.
3- Systolic or diatolic murmur on auscultation and thrilling on palpation of cardiac area.
4- Echocardiography reflects abnormal valve movement and

5- Grunting respiration from pain
6- Moderate ruminal tympany.
7- Scouring or constipation
8- Muscle weakness to the point of tremors and convulsion following by sudden death.
9- Distention of jugular vein and general oedema.
10- Emboli may originate and transfer to other organs, such as myocardium, kidney and joints.

Diagnosis

1- Case history
2- Clinical signs
3- Echocardiography
4- ECG: prolonged QRS complex indicating vulvular stenosis
5- Lab . Diagnosis:
   a. Leucocytosis.
   b. Increase in macrophages and monocyte.
   c. Severe anemia
   d. Hypergaammagobulinemia: leads to shortening of gluterhydecoagulation test (N: 3-5 minute).
   e. Blood culture to determine the causative MO.

Treatment

Treatment may not be effective because the thickness of the lesion prevent the access of antibiotics

1- Antibiotics in case of strept. Infection: procaine benzylpenicillin 20 000 iu/kg daily for 7-10 days. Relapse is common in endocarditis
2- If signs of congestive H.F. appear, the prognosis is poor.

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MYOCARDITIS

Definition

Myocarditis means inflammation of the hear muscles (myocardium) that is characterized by increase heart rate ans size in early stage and acute heart failure and syncope in late stage

Etiology

1- Bacterial myocarditis

a. Following bacteremia due to navel ill.
b. Clostridium chauvoei
c. Hemophilus somnus.

2- Viral myocarditis

- Foot and mouth disease (FMD) – especially young calves
- Viral leucosis in cattle

3- Parasitic myocarditis

- strongylus spp
- sarcocyst and neospora

4- Nutritioal deficiency

- Vitamin E and selenium deficiency
- chronic copper deficiency (falling disease)

5- chemicals:

- Drugs: catecholamines, xylazine, and monensin

6- Embolic infarction:

- Emboli from vegetative endocarditis

Inherited

- Congenital cardiomyopathy in Holstein cattle
- Glycogen storage disease: 1,4 glucosidase deficiency in shorthorn cattle

Clinical signs

1- In early stage: there is
   a. An increase in the heart rate and the heart size
   b. Tachyarrhythmia
   c. Changes in pulse and heart sound characters
2- In later or severe cases:
   a. Myocardial weakness may lead to hemic murmurs associated with the first heart sound and reach its peak with inspiration and disappear with expiration.
   b. Sudden death or attacks of cardiac syncope due to acute cardiac failure.
   c. Severe dyspnea or generalized edema due to congestive heart failure.
   d. ECG is of little value in assessing the state of myocardium but conduction disturbances may be detected.
   e. Echocardiography showed increased thickness of the wall of myocardium.

Diagnosis

1- Case history
2- Clinical signs
3- Differential diagnosis: acute heart failure. myocardial diseases is the major consideration in livestock with sudden death.
4- Lab. Diagnosis: muscle specific enzymes such as CPK and SGOT are elevated in the serum.

Treatment

1- avoid any excitement or exercise
2- treatment of the primary cause

3- injection of digoxin to increase muscle contraction, increase oxygen consumption increase the cardiac output and reduce the myocardial size: in cattle, digoxin is administered IV with dose of 2.2 mg/ 100 kg followed by 0.34 mg/kg every 4 hours.

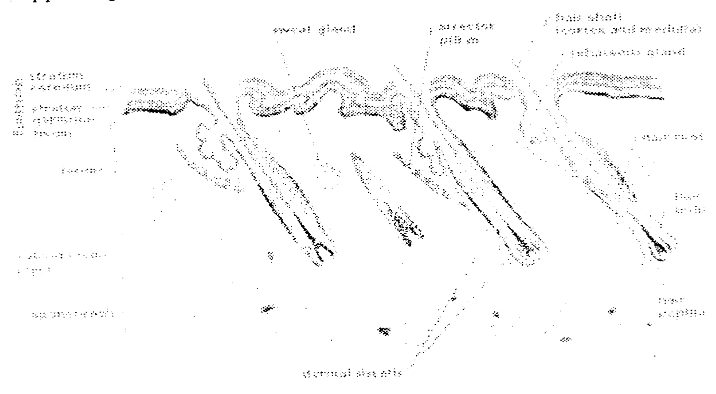
4- In more acute cases, where heart heart syncope occur, sudden death due to cardiac arrest or cardiac arrhythmia occur but intracardiac injection of very small dose of epinephrine could be helpful specially in ventricular fibrillation.

5- Broad spectrum antibiotic is given for prolonged. period. A combination of penicillin and gentamycin is valuable. Potentiated sulfonamides are also suggested

DISEASES OF SKIN

Introduction

The skin is the largest organ of the body and, depending on the species and age, may represent 12-24% of an animal's body weight. The skin has many functions, including serving as an enclosing barrier and providing environmental protection, regulating temperature, producing pigment and vitamin D, sensory perception, etc. Anatomically, the skin consists of the following structures: epidermis, basement membrane zone, dermis, appendageal system, and subcutaneous muscles and fat.



Principles of topical treatment

The following are some basic guidelines to consider when prescribing topical therapy.

1) As much of the hair coat as possible should be removed when treating skin diseases. Good grooming practices can significantly help shorten the course of disease. In addition, good grooming practices facilitate topical therapy.

2) The cooperation of the owner (and animal) should be evaluated before any topical therapy is prescribed.

3) Animals tend to groom off topicals. Local ointments, gels, and sprays are best used sparingly, under occlusion, and for specific diseases. Such medications often sting when applied to the skin, especially many of those instilled into the ears. Many agents also may mat the hair.

4) Tepid water is the temperature of choice for bathing animals.

5) The animal should be monitored closely for possible development of irritant or allergic contact dermatitis from topical agents. Many topical agents have very similar bases or ingredients, and changing from one to the other may just exacerbate the problem.

7) Owners should be given careful and thorough instructions on how to administer the therapy.

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PRURITIS (ITCHING)

Definition

This unpleasant sensation of irritation within the skin provokes the desire to scratch. It can be elicited only from the epidermis and palpebral conjunctiva. There are no specialized itch receptors. The itch sensation is carried from the nerve ending to the spinal cord and ascends the ventrolateral spinothalamic tract via the thalamus to the cortex

Etiology

1- Sarcoptic and chrioptic mange
2- Lice infestation
3- Nervous acetonemia
4- Early stage of photosensitive dermatitis
5- Urticarial wheals in allergic reaction
6- Itching of central origin may be caused by pseudorabies

Pathogenesis

1- The nature of the mediators of pruritus is controversial but is believed to include both histamines (released from mast cell degranulation) and proteolytic enzymes (proteases).
2- Proteases are released by fungi, bacteria, and mast cell degranulation, and during antigen-antibody reactions.
3- Leukotrienes, prostaglandins, and thromboxane A_2, which are broken down from arachidonic acid, are pro-inflammatory

Diagnosis

1- History
2- Clinical signs
3- Lab diagnosis

skin scrapings, Wood's lamp examination, fungal culture, allergy investigations (dietary tests, patch testing, and intradermal testing), and biopsy.

Treatment

1- Sedation of the animals
2- Anti-inflammatory corticosteroids: dexamethasone
3- Antibiotic: if pyoderma is suspected (Staph is the most common cause).

****

IMPETIGO

Definition:

It is a superficial eruption of tin-walled vesicles surrounded by a zone of erythema. The vesicles develop into pustules and rupture to form scabs.

Etiology:

1- The most common microorganism is staphylococcus
2- Udder impetigo due to staph. Infection.

Clinical signs

1- Vesicles appear chiefly on the relatively hairless parts of the body.
2- In the early stage, a zone of erythema is evident around the vesicle.
3- There is no irritation.
4- Rupture of the vesicle occurs rapidly or it may persist and becomes pustules which form yellow scabs.
5- Involvement of the hair follicle is common and leads to development of acne
6- Individual lesion may heal in a weak.



Udder impetigo in a cow

Treatment

1- Twice daily germicidal skin wash is usually adequate

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DERMATITIS

Definition: Dermatitis means inflammation of the skin including epidermis, dermis and may include blood vessels and lymphatics.

Etiology

1-Bacterial dermatitis: due to

a. Staphylococcus aureus (udder impetigo)

==

b. Corynebacterium pyogene

c. Streptococcus spp

d. Strawbery footrot and dermatophilus pedis .

2- Viral dermatitis: due to

a. Cowpox.

b. Foot and mouth disease (vesicles around natural orifices)

c. Ulcerative mammilitis.

d. Lumpy skin disease

e. Rinder pest, malignant cattharal fever (erosive lesion around natural orifices, eyes and coronets)

f. Vesicular stomatitis

3-Mycotic dermatitis: AS

a. Ring worm.

b. Dermatophilus congolensis

4-Metazoon dermatitis: As

a. Sarcoptic and psoroptic mange

5-Physical dermatitis: AS

a. Exposure to so cold or hot preparations

b. Photosensitization (primary or secondary)

6-Chemical dermatitis AS

a. due to strong acid as H_2SO_4

b. Arsenic preparation either local or parenteral with high concentration.

7-Nutritional dermatitis: zinc deficiency, vit A deficiency and vit B deficiency.

Clinical symptoms:

1- The affected skin area firstly show erythema and increase in the warmth

2- The subsequent stages vary according to the type and severity of the causative agent.

3- There may be development of discrete vesicular lesions or diffuse weeping.

4- Edema of skin and S/C tissue may occur in severe cases.

5- The next stage may be healing stage with scab formation or if the injuries are more severe there may be necrosis or even gangrene in the affected skin area.

6- Spread of infection to S/C tissue may leads to cellulitis or phlegmonous lesion or pyoderma (suppurative lesions).

7- Systemic reaction is likely to occur when the affected area is extensive.

8- Shock with peripheral circulatory failure, may be present with excessive burns.

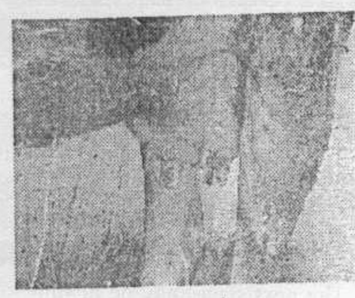
9- Toxaemia due to absortpion of tissue breakdown products or septicemia due to invasion via unprotected tissues may occur in later stage.

10- Bovine exfoliative dermatitis: occurs in calves due to excretion of unidentified agent in the dam's milk. The calves showed:

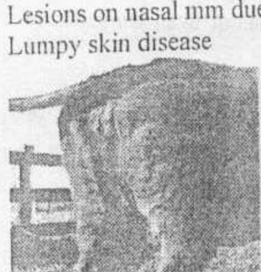
   a. Vesicles on the muzzle.
   b. Scaling and hair loss.
   c. The disease appears at a few days of age but recover spontaneously before 3 month.
   d. The dam has the same syndrome but mild and chronic form



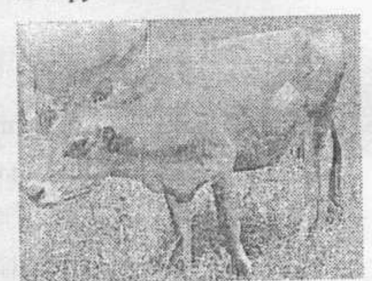
Lesions on nasal mm due to Lumpy skin disease



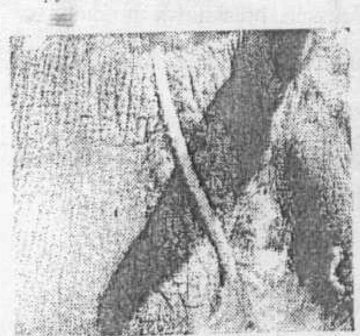
Lesions on teats due to Lumpy skin disease



Nodules on the back due to Lumpy skin disease



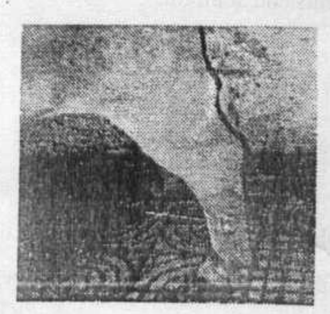
Nodules on the back due to lumpy skin disease



Parasitic dermatitis (sarcoptic mange)



Vesicles on the interdigital space due to FMD



Ulcer and edema in hind limb of a buffalo (bovine lymphangitis)



Mycotic dermatitis (ringworm)

Diagnosis:

1-Clinical features and history.

2-Skin biopsy and skin scraping to detect bacterial or parasitic dermatitis, respectively.

Treatment:

1- Removal of the offending cause.
2- Application of local antibiotics or antifungal.
3- Antihistaminic.
4- Anesthetic ointment when pain or itching is severe.
5- Providing of Vit. B in the food.
6- Astringent in weeping stage.
7- If shock present, fluid therapy should be administered.
8- Protein supplementation in diet for enhancing healing of skin.
9- Vaccination against viral causes as FMD.

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PHOTOSENSITIZATION

Definition: It is an acute dermatitis in non-pigmented areas of skin characterized by irritation, edema, sometimes anasarca (generalized edema) and necrosis of ears, eyelids, lips, face, vulva and cornet. It is

caused by sensitization of the superficial layer of lightly pigmented skin due to interaction of photodyamic pigments and sunlight.

Etiology and pathogenesis:

1-Primary photosensitization: due to

- Feeding on uncommon plants contain photodynamic pigment as saponine or mycotoxin.
- Chemicals and some drugs as phenothiazine (the metabolic end-product is phenothiazine sulfoxide is photosensitizing to calves). Cows treated with corticosteroids develop photosensitive dermatitis in udder and teats.

2- Secondary photosensitization (hepatogenous): hepatic diseases prevent the excretion of the phylloerythrin (metabolic end product of chlorophyll) leading to its accumulation and photosensitizing the skin.

Pathogenesis:

A- Normal metabolism of plant producing pigment in ruminant can be summarized as follow

1- Plant (contains chlorophyll) (in rumen by normal/ microbial action) → phylloerythrin → Absorption by portal circulation then to liver then reach the bile for excretion and consequently the peripheral blood circulation is free from phylloerythrin.
2- With liver disease → Generalized phylloerthrinemia.
3- Phylloerythrin in the skin absorb the sun light and activated (active form) in presence of protein molecules and amino acids and oxygen → it leads to the formation of toxic compounds and release of histamine → increase the permeability of capillaries to plasma proteins and consequently force the water into the affected tissue → edema and necrosis.

B- Molecules of photosensitizing agents (either primary or secondary) in the skin are energized by light. When the molecules return to the less energized state, the released energy is transferred to receptor molecules that quickly initiate chemical reactions in components of the skin. Tissue injury probably results from the production of reactive oxygen intermediates or from alterations in cell membrane permeability. Photosensitization differs from sunburn, in which lightly pigmented skin slowly becomes inflamed after exposure to ultraviolet rays.

4- Death due to secondary bacterial invasion, liver dysfunction, inability to feed and unthriftiness.

Clinical symptoms:

- The times from ingestion of plants contain photodynamic pigment and appearance of clinical signs varies from several hours to 10 days.
- The disease begins with

1- Erythema
2- Irritation of the lips, vulva, face, cornea, cornet and eyelids.
3- Rubbing and kicking of the affected parts (cows kicks affected teats).
4- Facial edema and in this stage there are:
   a. Thickening of lip and immobilization.
   b. Constricted and swollen nostrils.
   c. Dropping of heavy ears.
   d. Animal suffering from dyspnoea and anorexia
5- The skin disease may be severe enough to cause shock in the early stages.
6- Nervous signs including ataxia, posterior paralysis, blindness, and depression are observed

7- The animal that in convalescence may show dry gangrene and sloughing of the skin especially the ears.

8- Morbidity up to 80%, while mortality up to 20% of affected animals.

9- Icteric mucous membranes may occur.



Photosensitization (erythema) in a cow

P.M Lesions

1- Facial, vulvar, and pedal skin commonly is edematous.

2- Generalized icteric carcass as in case of secondary photosensitization.

3- Liver lesions may present as cirrhosis or bile obliteration.

Diagnosis:

1- Case history

a. Feeding on uncommonly plants

b. Non –pigmented areas

c. Administration of drugs as phenothiazine and corticosteroids.

2- Clinical findings and PM lesions.

3- Detection of the level of photodynamic pigment as phylloerythrin.

4- Differential diagnosis: either due to primary or secondary photosensitization.

5- Liver disease as aflatoxicosis, and liver abscesses
6- Other skin diseases.

Prevention and treatment:

1- Immediate removal from direct sunlight
2- Avoid grazing on pasture that containing photosensitizing agent.
3- Removing the plant containing photodynamic pigments from diet.
4- Rectal enema or laxative to remove toxic materials from affected animal.
5- Antihistamines as Avil, vetibenzamine and allercur.
6- Anti-inflammatory as dexamethasone, dexatimanol and finadin
7- Diuretic as lasix and sod citrate …etc.
8- Cold bath and palatable food.
9- Chronic liver disease rarely recovers.

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HYPERKERATOSIS

Definition: It is extensive keratinization of epithelial layer of the skin.

Etiology

1- Local: hyperkeratinization at pressure points, for example elbows when the animals lie habitually on hard surface.
2- Generalized: As in arsenic or napthalophethalin poisoning.
3- Deficiency of vitamin A.

Symptoms:

1- Area of alopecia and increase in the thickness of the skin.
2- Scale formation and dryness of the skin.
3- Cracks and fissures in grid-like fashion .
4- Secondary invaşion may occur.
5- Removal of hyperkeratotic material leaves intact underlying skin.

Diagnosis:

Clinical symptoms and skin scraping and biopsy

Treatment:

1- treatment of the primary condition
2- Washing of the skin by soap water.
3- Removing of the excessive keratinized layer by astringent as white lotion (zinc sulfate 2 gm + lead acetate 3 gm + 50ml D.W.).
4- The use of keratolytic agents, such as salicylic acid ointment may produce improvement.
5- Supplementation of vit. A.




Hyperkeratosis in a calf